
37th MD PhD
National Student Conference
July 8th-10th 2022

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WELCOME

Welcome to the 37th 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 Copper, 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.

We would like to thank our Keynote speakers – Dr. Craig Mello, Dr. Catherine Bollard, Dr. Peter Basser, Dr. Karl Deisseroth, & Dr. Yasmine Belkaid – for making the journey to share their work with us. We are also grateful to our career panelists and breakout session leaders for taking the time to lead discussions and share their insights with us.

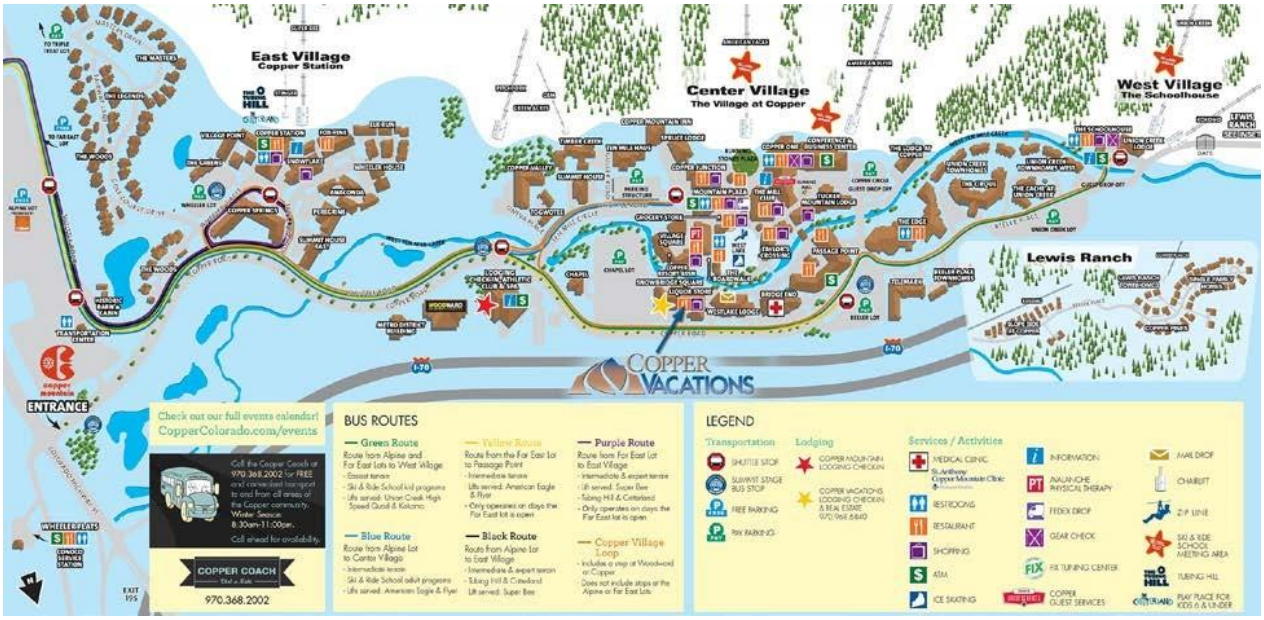
We hope that you gain insights into your own research and career as you participate in the scientific and career-oriented portions of the conference, as well as make certain to enjoy the surroundings of the Rocky Mountains. If you need anything during your time here, please do not hesitate to ask one of the conference organizers, identified by blue tie-dye shirts with the conference logo.

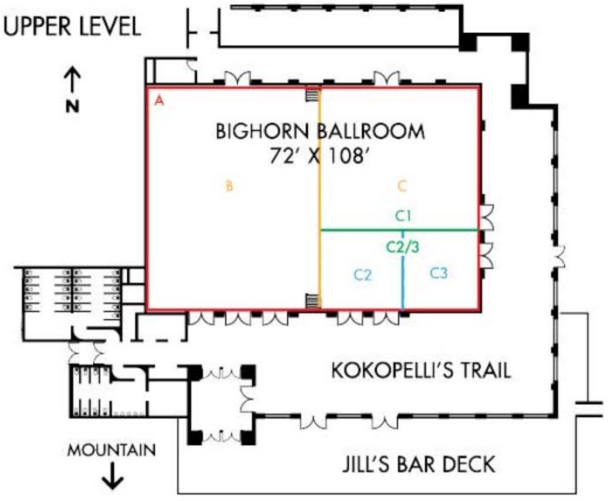
Sincerely,

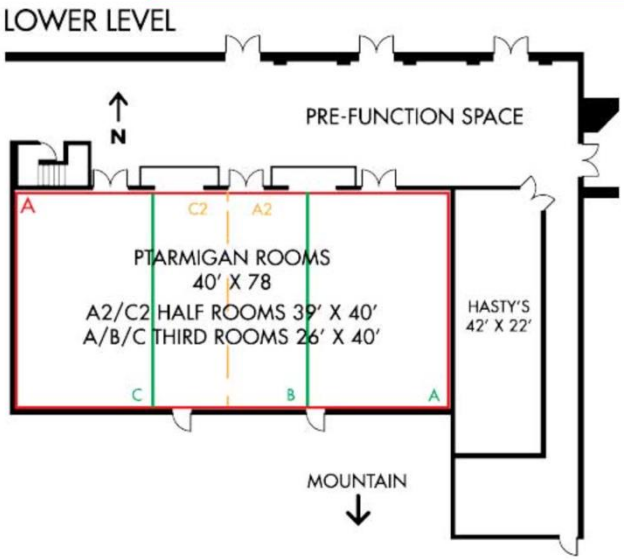
The 2022 Organizing Committee

Eric Barrientos
Chloe Briney
Keith Dodd
Nkolika Egbukichi
Mostafa El-Kalliny
Annika Gustafson
Jordan Hickman
Amita Kashyap
Uma Kantheti
Carley Miller
Varuna Nangia
Raquel Ortega
Dr. Daniel Sherbenou (Faculty Advisor)
Christopher Sienza (CU MSTP Administrator)

COPPER MOUNTAIN RESORT MAP







CONFERENCE SCHEDULE

All times listed in **Mountain Standard Time**

Friday, July 8

Time	Event	Room/Area
2:00 PM – 8:00 PM	General Registration Open	*Kokopelli's Trail*
5:30 PM – 7:00 PM	Dinner	*Bighorn Ballroom A*
6:00 PM – 7:00 PM	Keynote Speaker #1 **Dr. Yasmine Belkaid, PhD**	*Bighorn Ballroom A*
7:00 PM – 8:00 PM	Student Poster Session #1	*Kokopelli's Trail*
8:00 PM – 9:00 PM	Student Social Hours w/ Cash Bar	*Kokopelli's Trail*

Saturday, July 9

8:00 AM – 11:00 AM	Late Registration	*Kokopelli's Trail*
8:00 AM – 9:00 AM	Breakfast	*Bighorn Ballroom A*
9:00 AM – 10:00 AM	Student Oral Presentation Session #1	
	-Cancer Biology	*Ptarmigan A2*
	-Bioengineering & Structural Biology	*Ptarmigan C2*
10:00 AM – 10:15 AM	Morning Coffee Break	*Bighorn Ballroom A*
10:15 AM – 11:15 AM	Student Oral Presentation Session #2	
	-Neuroscience	*Ptarmigan A2*
	-Molecular & Cellular Biology	*Ptarmigan C2*
11:30 AM – 12:30 PM	Keynote Speaker #2 **Dr. Karl Deisseroth, PhD**	*Bighorn Ballroom A*
12:45 PM – 1:45 PM	Lunch & PSTP Director Panel	*Bighorn Ballroom A*
1:45 PM – 2:45 PM	Diversity, Equity, and Inclusion Panel	*Bighorn Ballroom A*
2:45 PM – 3:00 PM	Coffee & Snacks Break	*Bighorn Ballroom A*
3:00 PM – 4:00 PM	**PSTP Residency Showcases**	*Bighorn Ballroom A*
4:00 PM – 5:00 PM	Student Poster Session #2	*Kokopelli's Trail*
5:30 PM – 6:30 PM	Keynote Speaker #3 **Dr. Craig Mello, PhD**	*Bighorn Ballroom A*

Sunday, July 10

8:00 AM – 9:30 AM	Continental Breakfast Buffet	*Bighorn Ballroom A*
8:00 AM – 12:00 PM	Sunday Activities/Free Time: *Students and faculty can enjoy a mountain hike, go alpine fishing, or participate in Copper activities pass at an additional cost*	*Various locations*
12:00 PM – 1:30 PM	Lunch & Career Panel	*Bighorn Ballroom A*
1:30 PM – 2:30 PM	Keynote Speaker #4 **Dr. Peter Basser, PhD**	*Bighorn Ballroom A*
2:30 PM – 3:30 PM	Student Oral Presentation Session #3 -General Medicine I -General Medicine II	 *Ptarmigan A2* *Ptarmigan C2*
3:30 PM – 4:00 PM	Coffee & Snack Break	*Bighorn Ballroom A*
4:00 PM – 5:00 PM	Student Oral Presentation Session #4 -Immunology -Microbiology & Infectious Disease	 *Ptarmigan A2* *Ptarmigan C2*
5:00 PM – 6:00 PM	**Breakout Sessions** -Research Ethics -Scientific writing	 *Ptarmigan A2* *Ptarmigan C2*
6:00 – 6:30	Transition Break	
6:30 PM – 8:00 PM	Dinner and Keynote #5 **Dr. Catherine Bollard, MBChB, MD, FRACP, FRCPA**	*Bighorn Ballroom A*
8:00 PM	Closing Remarks & Awards	*Bighorn Ballroom A*

KEYNOTE SPEAKER I

Dr. Yasmine Belkaid, PhD

Distinguished Investigator, National Institute of Allergy & Infectious Diseases

Founder & Director, NIAID Microbiome Program

Co-director, Trans NIH Center for Human Immunology

Dr. Yasmine Belkaid is a Distinguished Investigator at the National Institute of Allergy and Infectious Diseases at the National Institute of Health (Bethesda). She obtained her Master's in Biochemistry at the University of Science and Technology Houari Boumediene in Algiers, Algeria, and her Ph.D. from Pasteur Institute in France. Following a postdoctoral fellowship at the National Institute of Health (Bethesda) on immune regulation during infection, she started her research program at the Children's Hospital Research Foundation in Cincinnati. In 2005, she joined the National Institute of Allergy and Infectious Diseases (NIAID) and was appointed senior scientist in 2008. Her laboratory explores fundamental mechanisms that regulate tissue homeostasis and host immune responses and uncovered key roles for the microbiota and dietary factors in the control of immunity and protection against pathogens. Dr. Belkaid is the co-director of the trans-NIH Center for Human immunology and is the founder and Director of the NIAID Microbiome program. Dr. Belkaid is a member of the National Academy of Sciences, the American Academy of Arts and Sciences, and the National Academy of Medicine and recipient of numerous awards including the Lurie Prize in Biomedical Sciences, and the Emil von Behring Prize, the Sanofi-Institute Pasteur Award, and the Robert Koch Award.

KEYNOTE SPEAKER II

Dr. Karl Deisseroth, MD PhD

Professor of Bioengineering and of Psychiatry & Behavioral Sciences at Stanford University

Karl Deisseroth is the D.H. Chen Professor of Bioengineering and of Psychiatry and Behavioral Sciences at Stanford University, and Investigator of the Howard Hughes Medical Institute. He received his undergraduate degree from Harvard, his PhD from Stanford, and his MD from Stanford. He also completed postdoctoral training, medical internship, and adult psychiatry residency at Stanford, and he is board-certified by the American Board of Psychiatry and Neurology. He continues as a practicing psychiatrist at Stanford with specialization in affective disorders and autism-spectrum disease, employing medications along with neural stimulation. Over the last sixteen years, his laboratory created and developed optogenetics, hydrogel-tissue chemistry (beginning with CLARITY), and a broad range of enabling methods. He also has employed his technologies to discover the neural cell types and connections that cause adaptive and maladaptive behaviors and has disseminated the technologies to thousands of laboratories around the world.

Among other honors, Deisseroth was the sole recipient for optogenetics of the 2010 Koetser Prize, the 2010 Nakasone Prize, the 2011 Alden Spencer Prize, the 2013 Richard Lounsbery Prize, the 2014 Dickson Prize in Science, the 2015 Keio Prize, the 2015 Lurie Prize, the 2015 Albany Prize, the 2015 Dickson Prize in Medicine, the 2017 Redelsheimer Prize, the 2017 Fresenius Prize, the 2017 NOMIS Distinguished Scientist Award, the 2018 Eisenberg Prize, the 2018 Kyoto Prize, and the 2020 Heineken Prize in Medicine from the Royal Netherlands Academy of Arts and Sciences. For his discoveries, Deisseroth has also received the Perl Prize (2012), the BRAIN prize (2013), the Pasarow Prize (2013), the Breakthrough Prize (2015) the BBVA Award (2016), the Massry Prize (2016) and the Harvey Prize from the Technion/Israel (2017). He was selected a Howard Hughes Medical Institute Investigator in 2013 and was elected to the US National Academy of Medicine in 2010, to the US National Academy of Sciences in 2012, and to the US National Academy of Engineering in 2019.

KEYNOTE SPEAKER III

Dr. Craig Mello, PhD

University Professor, University of Massachusetts Chan Medical School

Dr. Craig C. Mello received his B.Sc. degree in Biochemistry from Brown University in 1982 and received his Ph.D. from Harvard University in 1990. From 1990 to 1994 he conducted postdoctoral research at the Fred Hutchinson Cancer Research Center in Seattle, WA. He has been a member of the University of Massachusetts Chan Medical School faculty since 1995, and a Howard Hughes Medical Investigator since 2000. His pioneering research on RNAi, in collaboration with Dr. Andrew Fire, has been recognized with numerous awards culminating with the prestigious 2006 Nobel Prize in Physiology or Medicine.

KEYNOTE SPEAKER IV

Dr. Peter Basser, PhD

Senior Investigator, NIH

Director, Program on Pediatric Imaging and Tissue Sciences

Dr. Peter Basser received his A.B., S.M., and Ph.D. degrees in Engineering Sciences from Harvard University and his postdoctoral training in Bioengineering within the NIH IRP. In 1998, he became a Senior Investigator, and Chief of the new Section on Tissue Biophysics and Biomimetics (STBB), NICHD. From 2009 through 2015, he additionally served as Director of the Program on Pediatric Imaging and Tissue Sciences. He was then appointed to be the Associate Scientific Director for Imaging and Genomic Integrity within the NICHD IRP, a position he currently holds.

Dr. Basser is widely known for the invention, development, and clinical implementation of MR diffusion tensor imaging (DTI), diffusion tensor "streamline tractography," and other quantitative MRI methods for performing in vivo MRI histology or "microstructure imaging". These include CHARMED and AxCaliber MRI, which measure the mean axon diameter and axon diameter distribution, respectively, within white matter pathways, and various double Pulsed-Field Gradient (dPFG) or double wave-vector MRI methods, which are now widely used to elucidate distinct microstructural features of both gray and white matter in the brain. Within the area of neurotechnology, he made seminal contributions to our understanding of the physical underpinnings of transcranial magnetic stimulation (TMS) and its application in treating depression. He also wrote the first paper describing a new technique for delivering chemotherapeutic agents, which is now called "convection enhanced delivery" or CED.

Dr. Basser's notable awards and achievements include induction into the National Academy of Engineering (NAE), and receipt of the Gold Medal from the International Society of Magnetic Resonance in Medicine (ISMRM), as well as the Eduard Rhein Foundation Technology Award.

KEYNOTE SPEAKER V

Dr. Catherine Bollard, MBChB, MD, FRACP, FRCPA

Bosworth Chair for Cancer Biology
Director, Center for Cancer & Immunology Research
Professor of Pediatrics and of Microbiology, Immunology and Tropical Medicine
Director, Program for Cell Enhancement & Technologies for Immunotherapy
Children's National Health System
George Washington University School of Medicine and Health Sciences

Dr. Bollard received her medical degree at the University of Otago in Dunedin, New Zealand. She is board certified both in pediatrics and hematology. She worked in New Zealand and London, England, before moving to Houston, Texas, in 2000 where she was a Professor of Pediatrics, Medicine, and Immunology at Baylor College of Medicine (BCM) and the Director of the Texas Children's Cancer and Hematology Center Pediatric Lymphoma Program. In August 2013, she was recruited to Washington, DC, to join Children's National Health System and The George Washington University School of Medicine and Health Sciences.

Dr. Bollard is currently the Bosworth Chair for Cancer Biology, the Director of the Center for Cancer and Immunology Research, and the Director of the Program for Cell Enhancement and Technologies for Immunotherapy (CETI) at Children's National Health System. She is a Professor of Pediatrics and of Microbiology, Immunology, and Tropical Medicine at The George Washington University and the Associate Center Director for Translational Research and Innovation at the GW Cancer Center. Dr. Bollard is a member of the American Society for Clinical Investigation (ASCI), is a past president of the International Society for Cellular Therapy (ISCT) and is the current President of the Foundation for the Accreditation of Cellular Therapy (FACT).

Dr. Bollard was a member of the Cellular, Tissues, and Gene Therapies Advisory Committee of the Food and Drug Administration (FDA) from 2015 to 2019 and in 2019 she became a member of the Frederick National Laboratory Advisory Committee (FNLAC) for the NIH and an ad hoc member of the Pediatric Oncologic Drugs Advisory Committee (ODAC) for the FDA. She was an associate editor for the journal *Blood* from 2014-2021 and is currently Editor in Chief of *Blood Advances.* She has >200 peer reviewed publications and has been independently NIH funded for over a decade. Her bench and translational research focuses on improving outcomes for patients after hematopoietic stem cell transplantation and on the development of novel cell therapies for cancer and virus-associated diseases.

Residency Showcase & Panel

Saturday July 9th 3-4 PM, Bighorn Ballroom A

School	Program	Representative
Stanford University	Anesthesiology	Vivianne Tawfik, MD, PhD
Columbia University	Anesthesiology	Charles Emala, MD
Washington University School of Medicine in St. Louis	Clinical Pathology	Nima Mosammaparast, MD, PhD & Jacqueline Payton, MD, PhD
Vanderbilt University	Internal Medicine	Patrick Hu, MD, PhD
University of North Carolina at Chapel Hill	Internal Medicine	Joseph A. Duncan, MD, PhD
Harvard University	Internal Medicine	Jatin (Jay) Vyas, MD, PhD
University of Minnesota	Internal Medicine	Erik Peterson, MD
Stanford University	Internal Medicine	Joy Wu, MD, PhD
University of Wisconsin	Pediatrics	Emma Mohr, MD, PhD

Career Panelists

Sunday July 10th, 12-1:30 PM, Bighorn Ballroom A

Benjamin Young, MD, PhD

Dr. Benjamin Young is Head of Global Medical Directors of ViiV Healthcare where he supports clinical education, medical research and public health initiatives around the world. Since 2017, Dr. Young has been a principal of ViiV's Positive Perspective Study, one of the world's largest studies of outcomes reported by people living with HIV. From 2012 to 2018, Dr. Young was Senior Vice President and Chief Medical Officer of the International Association of Providers of AIDS Care, where he oversaw capacity-building programs and coordinated evidence-informed policies with the United Nations and the World Health Organization.

With over 20 years of experience in HIV medicine, Dr. Young was a Denver, Colorado-based clinician and clinical researcher. He was Co-Principal Investigator in the CDC HIV Outpatient Study from 1998 to 2018 and worked in Central Asia for two years as Head of Medical Affairs for Health Connections International, a Dutch non-governmental organization. Having authored more than 100 peer-reviewed scientific publications, Dr. Young has shared his HIV expertise through worldwide education programs where he has trained healthcare professionals and community groups in over 50 countries, with special focus in Eastern Europe, Central Asia and Mexico.

Dr. Young received his MD in 1992 and his PhD in Biochemistry and Molecular Biology in 1990 at the University of Colorado. He completed post-graduate training in Internal Medicine and Infectious Diseases at the University of Colorado Health Sciences Center in Denver.

Jatin Vyas, MD, PhD

Jatin (Jay) M. Vyas is an Associate Professor in Medicine at the Harvard Medical School and the Massachusetts General Hospital in the Department of Medicine, Division of Infectious Diseases, and an Associate Member of the Broad Institute. He attended the University of Texas at Austin in Plan II, a liberal arts honors program. He graduated with Special Honors from the University. From UT, he attended Baylor College of Medicine in their Medical Scientist Training Program where Jay received his PhD in 1994and MD in 1996.Upon completion of his MD, Jay moved to Boston to complete his medical internship and residency in internal medicine in the Department of Medicine at the Massachusetts General Hospital. He completed his fellowship in Infectious Diseases in the Massachusetts General Hospital/Brigham and Women's Hospital program. He then served as the chief resident in medicine for 18 months. Following a period of extended post-doctoral research training in the Harvard Department of Pathology and Whitehead Institute for Biomedical Research, Dr. Vyas joined the faculty of the Harvard Medical School and the Massachusetts General Hospital in 2007.Dr. Vyas is internationally recognized for

his work in fungal immunology, investigating the how our body responds to fungal pathogens. In 2014, Jatin was named the tenth Program Director of the MGH Department of Medicine Residency Program, supervising 207 interns and residents. As an NIH-funded investigator with interests in basic scientific discovery, Dr. Vyas provides a unique perspective to the medical house staff. He is married and has two children, aged 22 and 19 and resides in Milton, MA.

Lenny Dragone, MD, PhD

Leonard (Lenny) Dragone, MD PhD, is Chief Medical Officer at Sonoma Biotherapeutics. He joined Sonoma Bio from Janssen Biopharma where he served as the Vice President for Early Clinical Development as well as the interim Head of Data Sciences for Infectious Diseases. He was responsible for advancing therapeutic candidates from pre-IND enabling studies through Phase 1 to Phase 2b clinical trials as well as identifying high-impact data science studies. Lenny oversaw the development teams and created an infrastructure that bridged discovery to early development enabling collaboration and the creation of new patient-based studies to inform therapeutics in development for respiratory and chronic hepatitis B infections.

Lenny previously served as Senior Director of Experimental Medicine and Translational Pharmacology at Merck Research Laboratories in SSF. As clinical site lead and therapeutic area lead for Autoimmunity, Inflammation and Ophthalmology (AIO), he was responsible for early clinical development of the AIO pipeline through Phase 2a, contributing to pipeline strategy and prioritization. He also created a new experimental medicine clinical trials group functioning across the Merck network, which enabled experimental medicine studies across cardiovascular, metabolic, autoimmunity and inflammation indications. Prior to this, Lenny was a Medical Director in early clinical development at Genentech, leading and coordinating multiple cross-functional project teams with emphasis on IND filing and achieving proof-of-concept through Ph.2b for indications including SLE, RA, multiple sclerosis, psoriasis and influenza. Additionally, he created and ran an integrated clinical informatics team that worked at the interface of research, biomarker discovery and clinical investigation.

Lenny received his MD and PhD from the University of Rochester, before completing his Pediatric residency and Pediatric Rheumatology Fellowship training at University of California San Francisco (UCSF). He then ran his own NIH-funded laboratory publishing over 30 peer-reviewed publications and advanced up the academic ranks to Associate Professor of Pediatrics and Immunology at the University of Colorado and National Jewish Health in Denver Colorado. Lenny continues to stay clinically active as a Volunteer Associate Professor of Pediatric Rheumatology at UCSF seeing patients in the pediatric rheumatology fellow's clinic.

Craig Forester, MD, PhD

Craig Forester, MD, PhD is an assistant professor at the University of Colorado-Anschutz and Children's Hospital of Colorado. He completed his MD, PhD at the University of Utah before heading to Boston Children's Hospital/Boston Medical Center for training in Pediatrics where he became interested pediatric bone marrow failure. He then moved to the University of California-San Francisco for fellowship training in Pediatric Hematology/Oncology/BMT where he developed techniques to study nascent proteomics and translational control in early hematopoiesis in the lab of Davide Ruggero. Currently, at the University of Colorado, Dr. Forester studies the intersection between epitranscriptomics and RNA structure in translation specificity and attends in Pediatric Hematology.

Dr. Brianne Barker, PhD

Dr. Brianne Barker is an Associate Professor of Biology at Drew University, where she has taught undergraduate courses and performed research since 2013. Prior to her work at Drew, Dr. Barker earned a B.S. in Biology from Duke University in 2002 and a Ph.D. in Immunology from Harvard University, where she examined DNA vaccine-elicited immune responses against HIV and CD8 T cell memory. She then completed postdoctoral training at the University of North Carolina at Chapel Hill, examining innate immune responses in bacterial, viral, and inflammatory diseases. Dr. Barker's teaching interests focus on Immunology, Virology, Emerging Infectious Disease, and Microbiology. Her research laboratory focuses on understanding innate immune responses to viral infection and vaccines. Dr. Barker also applies her science and education training to science communication, particularly throughout the pandemic, working with the American Society for Virology on various pandemic communication initiatives and co-hosting two podcasts: "This Week in Virology" and "Immune".

Breakout Sessions

Sunday July 10th, 5-6 PM

Research Ethics (Ptarmigan A2)

Led by Mary Allen, PhD - "Surviving the ethical swamps of scientific research"

Dr. Allen is a Research Assistant Professor and RCR coordinator at the University of Colorado Boulder. Her current research revolves around transcription factor function in Trisomy 21, and her lab uses techniques that range from molecular biology to machine learning. When Dr. Allen was a graduate student, the graduate student in her lab discovered likely falsification of data in grants submitted by their PI. This event was a turning point in Dr. Allen's career. Dr. Allen will speak of her views on responsible conduct of research before, during, and after this event. Dr. Allen, and the other students in her grad school lab, have been written about in several books on research misconduct, and RCR instructors often use their situation as a case study in the training of students in RCR.

Scientific Writing (Ptarmigan C2)

Led by Brianne Barker, PhD

Dr. Brianne Barker is an Associate Professor of Biology at Drew University, where she has taught undergraduate courses and performed research since 2013. Prior to her work at Drew, Dr. Barker earned a B.S. in Biology from Duke University in 2002 and a Ph.D. in Immunology from Harvard University, where she examined DNA vaccine-elicited immune responses against HIV and CD8 T cell memory. She then completed postdoctoral training at the University of North Carolina at Chapel Hill, examining innate immune responses in bacterial, viral, and inflammatory diseases. Dr. Barker's teaching interests focus on Immunology, Virology, Emerging Infectious Disease, and Microbiology. Her research laboratory focuses on understanding innate immune responses to viral infection and vaccines. Dr. Barker also applies her science and education training to science communication, particularly throughout the pandemic, working with the American Society for Virology on various pandemic communication initiatives and co-hosting two podcasts: "This Week in Virology" and "Immune".

Diversity, Equity, and Inclusion Panel

Saturday July 9th, 1:45-2:45 PM, Bighorn Ballroom A

Talia Swartz, MD, PhD

Talia H. Swartz, MD, PhD, is Associate Professor in the Department of Medicine, Division of Infectious Diseases and Medical Education. She is a physician scientist, MD-PhD graduate from the Mount Sinai MSTP in 2008. She completed internal medicine research track residency at the Mount Sinai Hospital and then Infectious Diseases fellowship at the Mount Sinai hospital. She joined the laboratory of Benjamin Chen studying the biology that underlies chronic HIV infection which led to a mentored career award (K08) and current runs a research laboratory that investigates the signaling that mediates inflammatory signaling in response to infection and aims to develop novel treatment paradigms to reduce the morbidity and mortality of HIV-1 disease and COVID-19. Her clinical practice is focused on care for people with HIV and, more recently, COVID-19. Dr. Swartz is Co-Director of the MD/PhD Program and Associate Dean for MD/PhD Education. She has been deeply committed to supporting the training of physician-scientists through the recruitment and support of diverse trainees who are navigating dual training in medicine and biomedical science. She is involved in innovative curricular development, outreach, and student support, both at the Icahn School of Medicine at Mount Sinai and through the AAMC GREAT Group MD/PhD Section Communications Committee where she has been developing resources to make physician-scientist training available to a more diverse group of future trainees. She is also Director of Graduate Research and Education of the new Center for Antiracism in Practice at Mount Sinai, which is serving to identify the insidious and pervasive nature that structural racism impacts graduate education and is working to form a Community of Practice to dismantle racism and create a more inclusive culture for physician-scientist training.

Tarik Walker, MD, MPH

Dr. Tarik Walker, M.D., M.P.H., currently is a Physician-Scientist at the University of Colorado. Dr. Walker is a medical graduate from Johns Hopkins University Medical School and Public Health School. He has also conducted health disparities research at the UC Hospital as an Infectious Disease/HIV Research Fellow. Prior to his recent appointment at the University of Colorado, Dr. Walker conducted quantitative and qualitative research investigating perceptions of the Health Professions by inner-city high school youth at the Medical College of Wisconsin as a Pediatric fellow. His research assessing minority high school medical interests and perceptions of the Health Professions is one of his many research interests. Dr. Walker was also trained as a LEND Fellow in areas of disease prevention and intervention and advocacy for investigating pediatric autism and developmental disabilities in underserved communities as it relates to health disparities. As an African-American researcher invested in underserved communities here in Denver/Aurora, Dr. Walker serves as both an academic resource as well as a community liaison.

In addition to leadership and investigative work, he is also passionate about continuing his work as an instructor, while mentoring the next generation of (underserved) scientists/physician-scientists. Dr. Walker now seeks to leverage his background as a recent Strategic Initiatives and Health Equity Officer within El Paso County Public Health by working

to prioritize and address the health needs of historically underserved citizens/communities within Colorado.

Olujimi A. Ajijola, MD, PhD

Associate Professor of Medicine

Director, UCLA Neurocardiology Research Program

Co-Director, UCLA Caltech Medical Scientist Training Program

Associate Director, Specialty Training and Advanced Research (STAR) Program

UCLA Cardiac Arrhythmia Center, David Geffen School of Medicine at UCLA

Dr. Ajijola completed his undergraduate studies at the University of Virginia and received his medical degree from Duke University. He went on to the Massachusetts General Hospital for residency training in internal medicine and completed clinical fellowships in cardiovascular medicine and cardiac electrophysiology at UCLA. He received a Ph.D. in Molecular, Cellular, and Integrative Physiology at UCLA, as part of the Specialty Training and Advanced Research (STAR) program. He is interested in novel approaches for cardiac arrhythmias and performs invasive cardiac electrophysiological procedures. In addition to the NIH Director's New Innovator award, he is a recipient of the Jeremiah Stamler Cardiovascular Research Award, an A. P. Giannini Foundation post-doctoral award, and a Young Physician Scientist Award from the American Society for Clinical Investigation (ASCI). He is a member of the New Voices program of the National Academies of Science, Engineering, and Medicine. He directs the Neurocardiology Research Program and Clinical Autonomic Testing Laboratory at UCLA. He co-directs the NIH-funded UCLA-Caltech Medical Scientist Training Program, serves on the leadership team of the UCLA Specialty Training and Advanced Research (STAR) program, and directs the UCLA internal medicine residency physician-scientist training program (ProSTAR-PSTP).

Yentli Soto Albrecht

Yentli Soto Albrecht is a 4th year MD-PhD student at the University of Pennsylvania Perelman School of Medicine (Penn). She earned a degree in Molecular Biology at Princeton University, and worked at the Ragon Institute of MGH, MIT, and Harvard before coming to Penn. She is undertaking her thesis in the Douglas Wallace lab at the Children's Hospital of Philadelphia, where she studies the role of mitochondria in SARS-CoV-2 pathogenesis. Specifically, she studies aspects of mitochondrial function that serve to limit SARS-CoV-2 replication and how mitochondrial background may contribute to COVID-19 severity. She is rising president of the American Physician Scientists Association, where she enjoys working with a passionate team to support other trainees and increase the diversity of the physician-scientist workforce. She hopes to fight emerging viral infections globally with science and medicine throughout her physician-scientist career.

STUDENT ORAL PRESENTATION SCHEDULE

Session 1, Saturday July 9th, 9:00-10:00 AM

I. Cancer Biology (Ptarmigan A2)

9:00-9:15 AM: Mary Bedard, University of Cincinnati, A single cell transcriptome atlas of the HPV16-infected squamous epithelium identifies a novel epithelial subpopulation implicated in cancer

9:15-9:30 AM: Anthony Restaino, University of South Dakota, Electro-physiologically active tumor microenvironment promotes cancer progression

9:30-9:45 AM: Francisco Neal, University of Texas Health San Antonio, A tumor suppressor BRCA2 safeguards genome integrity through two distinct DNA binding domains

9:45-10:00 AM: Kyra T. Newmaster & Scott Hwang, Penn State, Predicting CNS Tumor mmp9 Methylation Status Using MRI Features

II. Bioengineering & Structural Biology (Ptarmigan C2)

9:00-9:15 AM: Oluwamayokun Oshinowo, Emory University, Single platelet force as a biophysical biomarker for clinical bleeding severity

9:15-9:30 AM: Bojing Blair Jia, University of California San Diego, A spatial genome aligner for multiplexed DNA-FISH

9:30-9:45 AM: Kaylie Cullison, University of Miami, Weekly Dynamics of Glioblastoma drug Treatment on MRI-Linac

9:45-10:00 AM: Jordan E. Morningstar, Medical University of South Carolina, Mitral valve prolapse induces regionalized left ventricular fibrosis

Session 2, Saturday July 9th, 10:15-11:15 AM

III. Neuroscience (Ptarmigan A2)

10:15-10:30 AM: Lincoln Wurtz, Mayo Clinic, The Dynamic Role of Kallikrein 6 in Myelination

10:30-10:45: Sarah Zych, University of Colorado, Divergent properties and independent regulation of striatal dopamine and GABA co-transmission

10:45-11:00: Tyrone DeSpenza, Yale School of Medicine, Therapeutic targeting of mTOR in a genetic subtype of congenital hydrocephalus

11:00-11:15: Akansha Jain, University of Iowa, Modulation of cellular ATP levels as a potential therapeutic strategy for Parkinson's disease.

IV. Molecular & Cellular Biology (Ptarmigan C2)

10:15-10:30 AM: Andrea Toth, University of Cincinnati, Understanding and preventing perinatal lung injury in a rhesus macaque model of chorioamnionitis

10:30-10:45: Kelsey Nolden, Medical College of Wisconsin, A novel Fis1 inhibitor reverses diabetic endothelial dysfunction in human resistance arteries

10:45-11:00: Juan Colazo, Vanderbilt University, Albumin-hitchhiking MMP13 siRNA (siMMP13<(EG18L)2) for the treatment of Rheumatic Disease

11:00-11:15: Javier Sierra Pagan, University of Minnesota, ETV2 functions as a pioneer transcription factor that regulates endothelial lineage development and reprogramming

Session 3, Sunday July 10th, 2:30-3:30 PM

V. General Medicine I (Ptarmigan A2)

2:30-2:45 PM: Havell Markus, Pennsylvania State College of Medicine, Utilizing electronic health records to identify high risk ANA-positive patients that may progress to systemic lupus erythematosus

2:45-3:00 PM: Simone Herzberg, Vanderbilt University, Obesity and Rotator Cuff Disease: A Systematic Review and Meta-Analysis

3:00-3:15 PM: Al Smith, Carle Illinois College of Medicine, Quantifying the Need for Global Emergency Neurosurgical Intervention

3:15-3:30 PM: Lily Nguyen, University of Colorado, Combination epigenetic and PARP inhibitor therapies can uncover transposable elements with gene regulatory function in ovarian cancer cells

VI. General Medicine II (Ptarmigan C2)

2:30-2:45 PM: Jared Beyersdorf, Emory University/Georgia Institute of Technology, Robust, Durable Gene Activation In Vivo via mRNA-Encoded Activators

2:45-3:00 PM: Mary Adeyeye, University of Texas, Paradoxical Effect of Frizzled2-Fc on Bone Mass for the Treatment of Osteogenesis Imperfecta

3:00-3:15 PM: Maryknoll Linscott, Pennsylvania State College of Medicine, PIK3CA and cMYC promote the expansion of distinct Ras-initiated, long-lived premalignant clones in a multistage murine breast cancer model

3:15-3:30 PM: Kyle Woisard, Virginia Commonwealth University, Executive Control Network Resting State fMRI Functional Connectivity in Cocaine Dependent Subjects

Session 4, Sunday July 10th, 4:00-5:00 PM

VII. Immunology (Ptarmigan A2)

4:00-4:15 PM: Jacqueline Turner, University of Colorado, Lysophosphatidic acid rewires CD8 T cell metabolism and anti-tumor immunity

4:15-4:30 PM: Peter Dimitrion, Wayne State University, Dissecting peripheral and cutaneous immunological dysregulation in patients with hidradenitis suppurativa

4:30-4:45 PM: Sara Bolivar Wagers, University of Minnesota, Prevention of acute graft-versus-host disease using an orthogonal IL-2/IL-2Rβ system to selectively expand regulatory T cells in vivo

4:45-5:00 PM: Jonathan Liang, Yale School of Medicine, Saturated fatty acid activation of the NLRP3 inflammasome depends more strongly on JNK activation than on ROS

VII. Microbiology and Infectious Diseases (Ptarmigan C2)

4:00-4:15 PM: Yentli E. Soto Albrecht, University of Pennsylvania, Non-pathogenic variation in mitochondrial DNA modulates murine SARS-CoV-2 pathogenesis

4:15-4:30 PM: Umaru Barrie, University of Texas Southwestern, ERK1/2 Activation in Macrophages is Necessary for Efficient Leishmania amazonensis Internalization and Pathogenesis

4:30-4:45 PM: Soo hun Yoon, Michigan State University, Uncovering the role of ubiquitin-like modulation of 3'3'-cGAMP signaling in Vibrio cholerae El tor

4:45-5:00 PM: Hannah Bell, University of Rochester, The Function of Type Three Secretion System Protein VopZZ

ORAL PRESENTATION ABSTRACTS

Session 1: Cancer Biology

Mary Bedard

University of Cincinnati

A single cell transcriptome atlas of the HPV16-infected squamous epithelium identifies a novel epithelial subpopulation implicated in cancer

Persistent HPV16 infection is a major cause of the global cancer burden. The viral life cycle is dependent on the differentiation program of stratified squamous epithelium, but the landscape of keratinocyte subpopulations which support distinct phases of the viral life cycle has yet to be described. Single cell RNA sequencing of HPV16 infected versus uninfected organoids identified twelve keratinocyte populations representing distinct epithelial subpopulations: four basal (C4, C5, C7, C10), one proliferating (C6), six differentiated (C0-3, C8, and C11), and one unique cluster divergent from the known squamous differentiation program that was termed differentiation-discordant (C9). Subpopulations unique to HPV16+ tissue were defined (C8) as well as subpopulations highly enriched in HPV16+ epithelium (C6, C9). Cluster-defining genes were validated as biomarkers for select epithelial subpopulations and used to map distinct epithelial subpopulations spatially in 3D rafts. Instead of conventional terminally differentiated cells, an HPV-reprogrammed keratinocyte subpopulation (C9) formed the new surface compartment of HPV+ rafts. C9 biomarkers (e.g. ELF3 and GPR110) were not detected in normal human epidermis or cervical tissue, but persisted through stages of human carcinogenesis. Specifically, a C9 compartment was detected in HPV+, but not HPV–, head and neck squamous cell carcinomas and superjacent to p16+ regions of cervical intraepithelial neoplasias. A role in promoting viral carcinogenesis was further supported by strong association of the ELF3 transcription factor with HPV+ human cancers in the TCGA. Importantly, ELF3 knockdown abolished this unique cellular compartment in HPV16+ epithelial rafts. Altogether, we report the first single cell transcriptome atlas of HPV16-infected epithelium and identify unique cell populations harboring distinct biomarkers and molecular networks. The newly discovered cell populations and signaling molecules may be viral targets with potential utility for the rationale design of novel diagnostic, prognostic, and therapeutic strategies for HPV-associated cancers. This transcriptome atlas will enable broader studies of the role of individual keratinocyte subpopulations in tumor virus infection and cancer evolution.

Anthony Restaino

University of South Dakota

Electro-physiologically active tumor microenvironment promotes cancer progression

The presence of intra-tumoral neurons in peripheral cancers is now widely accepted. Intra-tumoral neurons are recruited by various processes and the neuronal density correlates with poor prognosis. Despite this, the electro-physiological activity (EPA) of intra-tumoral neurons and how it contributes to cancer progression, is poorly understood.

Using micro-electrode arrays, we demonstrate a significant increase in recorded EPA from HNSCC samples compared to normal tonsil controls. Since HNSCCs encompass two diseases, human papillomavirus (HPV+) driven and mutationally driven (HPV-), we separated samples based on HPV status. We found that HPV- tumors harbor significantly elevated EPA compared to HPV+ tumors. Immunohistochemical staining for the neuron marker β-III tubulin shows this EPA

difference reflects the difference of neural densities between the groups. Consistent with this, tumors from TRPV1-DTA transgenic mice harbor significantly decreased electrical activity. Proximity ligation assays demonstrate increased synaptic units in HPV- compared to HPV+ tumors and normal tissue controls. Nerve tracing indicates that intra-tumoral neurons originate from ipsilateral trigeminal ganglia (TG), and tumor presence promotes increased substance P (SP). Importantly, human and mouse HNSCC cell lines express the SP receptor NK1R. SP treatment of human and mouse HNSCC cell lines increases their proliferation and migration, which is inhibited by treatment with an antagonist to NK1R. Western blot analysis shows that SP binding of NK1R on tumor cells increases signaling through the MAP Kinase pathway. Here, we show that intra-tumoral neurons promote elevated EPA in the tumor environment. These neural structures establish functional synapse-like structures via neurons recruited from the TG and have increased expression of SP. Neuronal release of SP engages tumor cell expressed NK1R and drives cellular proliferation and migration mediated through activation of the MAP Kinase pathway.

Francisco Neal

University of Texas Health San Antonio

A tumor suppressor BRCA2 safeguards genome integrity through two distinct DNA binding domains

Damage to the genome can cause genetic mutations and chromosome rearrangements capable of driving neoplastic cell transformation and oncogenesis. Cells have evolved a host of specialized DNA repair pathways to eliminate potentially deleterious DNA lesions. One such pathway is homologous recombination (HR), a high-fidelity process used to repair DNA double-strand breaks (DSBs), interstrand crosslinks, and collapsed DNA replication forks. Proper execution of HR requires the tumor suppressor BRCA2 (Breast Cancer 2). Germline mutations in BRCA2 lead to breast and ovarian cancer, as well as the cancer-prone syndrome Fanconi anemia. We have shown that, in addition to its OB-fold rich DNA-binding domain (DBD), BRCA2 possesses a distinct DNA binding activity in the C-terminal recombinase binding domain (CTRB). We have yet to determine how these two domains contribute to BRCA2-mediated DNA break repair and stalled replication fork protection. To address this knowledge gap, we have utilized a combination of genetic and biochemical approaches to delineate how the DNA-binding activity of the DBD and CTRB guide BRCA2-mediated HR repair and maintain stability of stressed replication forks. We have found that the CTRB binds DNA with two distinct patches of amino acid residues, and loss of this DNA-binding activity adversely impacts upon the ability of BRCA2 to function as a recombination mediator that facilitates RAD51-ssDNA filament assembly. Furthermore, we have found that mutation of basic residues in the second and third OB folds of the DBD impairs binding of ssDNA and partly abrogates recombination mediator activity. Our results thus reveal a novel DNA binding attribute in the BRCA2 CTRB and clarify the role of the DBD and CTRB in homologous recombination and replication fork preservation. Knowledge of these domains will lay a foundation for efficient identification of cancer-associated mutations and development of novel therapeutics to treat BRCA2-mutated cancers.

Kyra T. Newmaster & Scott Hwang

Pennsylvania State College of Medicine

Predicting CNS Tumor mmp9 Methylation Status Using MRI Features

Gliomas remain one of the most difficult to treat cancers with the mainstay being resection followed by radiation. However, treatment regimens can include bevacizumab which reduces the production of new blood vessels, a prerequisite for the growth and spread of cancer. However, objectively identifying candidates with high response probability is difficult. One possible indication for the use of bevacizumab is high matrix metalloprotease 9 (MMP9) expression within the tumor which is directly controlled by methylation of the MMP9 gene. Therefore, we wanted to predict MMP9

methylation status based on imaging features rather than relying on tumor biopsy and sequencing. To answer this question, we selected samples from the TCGA Low grade glioma and glioblastoma databases which had genetics, imaging, and clinical data available (N=128). First, we confirmed that MMP9 hypomethylation correlated with increased MMP9 mRNA (average level=4.6 (n=77) vs 1.3 (n=45), p<0.0001) and decreased survival (n=57 (hypomethylated) vs 44 (methylated), Kaplan-Meier, p=8.53e-10). Then, we used 482 different volumetric, intensity, microstructural, and textural MRI features from 89 MRI image sets to train an XGBoost model to predict tumor MMP9 methylation status. The remaining 39 samples were used to test the prediction capability of this model which yielded an F1 score of 0.82. Subsequent SHAP analysis showed that only 29 features including 15 texture, 3 volumetric, and 6 histological measures made a significant impact on the model, and when these 30 features were used to train a new model (n=89), we achieved an F1 value of 0.84 using a test set containing 39 samples. In conclusion, we generated a machine learning model that only utilizes 29 features to predict MMP9 methylation status of tumors based on imaging alone which provides a non-invasive technique for predicting patients that are more likely to respond to bevacizumab treatment.

Session 1: Bioengineering & Structural Biology

Oluwamayokun Oshinowo

Emory University

Single platelet force as a biophysical biomarker for clinical bleeding severity

Immune Thrombocytopenia Purpura (ITP) is a bleeding disorder characterized by low platelet counts. While most of the 4,000 new cases of pediatric ITP diagnosed each year self-resolve, 10% of these patients experience major bleeding episodes, and 0.5% suffer life-threatening intracranial hemorrhage. The platelet count remains the mainstay method for predicting hemorrhage and unfortunately only loosely correlates to bleeding severity. Consequently, there currently exists no biomarker that accurately and reliably predicts which patients need immediate medical treatment and which patients only require monitoring. One key barrier in assessing platelet function in ITP patients is the low platelet count which adversely affects and confounds commonly used assays of platelet function. To that end, we have developed a new technology, the Platelet Contraction Cytometer (PCC), that simultaneously measures the contractile force of hundreds of individual platelets, to study platelets of patients with ITP prospectively. Buchanan bleeding scores were used to distinguish patients with severe symptoms from asymptomatic patients. With 49 patients, we observed three significant findings: 1) Tracking both single platelet force measurements and platelet count enables stratifying patients into major, minor, or minimal risk for bleeding. Accordingly, patients in the major risk category have a combined low platelet force and low platelet count. 2) Longitudinal studies showed that when major risk patients had increased platelet force or higher platelet counts, this was associated with the alleviation of major symptoms and 3) importantly, the mechanism behind the decreased platelet force is immuno-mechanically modulated by a patient's own antibodies toward the important contraction integrin GPIIb/IIIa. Thus, our data suggests that platelet forces may represent a new category of clinical diagnostic biomarker– one that is biophysical in nature and is modulated by platelet reactive antibodies.

Bojing Blair Jia

University of California San Diego

A spatial genome aligner for multiplexed DNA-FISH

Multiplexed fluorescence in situ hybridization (FISH) has emerged as a powerful approach for analyzing 3D genome organization, but it is eminently challenging to derive chromosomal conformations from noisy fluorescence signals. Tracing chromatin is not straightforward as chromosomes lack conserved shapes for reference checking whether an observed fluorescence signal belongs to a chromatin fiber or not. Here we report a spatial genome aligner that parses true chromatin signal from noise by aligning signals to a DNA polymer model. We demonstrate that this spatial genome aligner can efficiently reconstruct chromosome architectures from DNA-FISH data across multiple scales (5kb, 25kb, 1Mb). Further, it can parse tightly intertwined sister chromatids otherwise inseparable by eye, and determine chromosome ploidies de novo in interphase cells. Reprocessing of previous whole-genome mouse chromosome tracing data with this method revealed a distinct population of cells lacking two chromosome territories and instead the spatial compaction of different homologs into one chromosome territory in S/G2 mouse embryonic stem cells (mESCs). We further show within these compact territories, sister chromatids of different homologs lose pairing towards their attendant sister and may pair with sisters of the other homolog, a structure evocative of mitotic cross-over. Finally, we uncovered extranumerary chromosomes that tightly pair with another chromatin fiber, a structure evocative of sister chromatids, in post-mitotic neurons of the adult mouse cortex. Our spatial genome aligner may facilitate the adaption of multiplexed DNA-FISH by the community.

Kaylie Cullison

University of Miami

Weekly Dynamics of Glioblastoma drug Treatment on MRI-Linac

Introduction: Few studies have investigated glioblastoma (GBM) changes during chemoradiotherapy (chemoRT). Many tumors grow and many post-operative resection cavities (RC) shrink when comparing pre- and post-treatment MRI. MRI-linac, combination MR-RT devices, offer the opportunity to investigate tumor dynamics during chemoRT without standalone MRI. The purpose of this study was to investigate intracranial changes during chemoRT using imaging from MRI-linac for future prospective trials. Methods: Using an IRB-approved prospective cohort of 34 GBM patients undergoing 30 treatments of chemoRT to 60 Gy on a 0.35T MRI-linac, tumor/edema (lesion) and RC dynamics throughout treatment were analyzed on T2-weighted treatment set-up scans. RC/lesion change was measured for weekly time points (W1-W6). Absolute and relative volume (>1 indicates growth, <1 indicates shrinkage) changes compared to the pre-treatment planning scan (PL) and migration distance, the largest linear distance that RC/lesion migrated from PL, were calculated. Target RT margins required through treatment to avoid lesion growth out of margins and how much normal brain can be saved by adapting RT margins to shrinking RC were investigated. Results: 8 patients had RC only, 10 had lesion only, and 16 had both RC and lesion. In general, resection cavities shrunk, while lesions grew during treatment. The treatment margin required to avoid the average lesion from growing out of margins at any point during chemoRT from PL (i.e., migration distance) is 1.30 cm (max 4.1 cm). Adapting RT margins to a shrinking RC (RC only or RC with small lesion <20 ml (n=11)), assuming a 2 cm expansion of lesion for the clinical target volume adapted at W5, saves 26.92 ml (range 9.66 – 58.63 ml) of normal appearing brain from full RT dose. Conclusion: Clinically significant anatomic changes were seen in GBM patients during chemoRT. Patients with unresected lesions require large RT margins or volume expansions for growth during RT. As RCs shrink, treatment margins can be reduced to save normal brain. GBM trials should strongly consider adaptive RT given the significant GBM variability during treatment.

Jordan E. Morningstar

Medical University of South Carolina

Mitral valve prolapse induces regionalized left ventricular fibrosis

Recent imaging studies in patients with mitral valve prolapse (MVP) have shown cross-sectional evidence of regionalized left ventricular (LV) fibrosis as well as increased risk for sudden cardiac death. How fibrosis develops in MVP is unknown, however, the distribution of fibrosis led us to hypothesize that direct mechanical tension as a result of a billowing leaflet may drive fibrosis. To test our hypothesis, we performed histopathologic analysis on cardiac biopsies of 6 patients who underwent surgical repair for MV prolapse. We additionally performed longitudinal histopathologic analysis on hearts harvested from a mouse modal of human non-syndromic MVP (Dzip1S14R/+). Finally, to assess the cellular response to increased mechanical tension, pathological stretch assays followed by RNA Seq was performed on primary human cardiac fibroblasts. We found that human endomyocardial biopsies from LV inferobasal wall, LV septum, and LV apex demonstrated regionalized myocardial fibrosis that correlated with increased macrophages and myofibroblasts. Our mouse model demonstrated similar regionalized fibrosis that increased over time, as well as an increase in macrophages and myofibroblasts. Finally, direct mechanical stretching of cells pushed fibroblasts into a pro-fibrotic state, and significantly altered expression levels of 232 genes, including genes known to play a role in fibrosis such as ACTA2, COL1A1 and COL1A2, LOX, and PLOD1. Collectively our findings demonstrate a direct mechanistic role for increased mechanical tension due to leaflet prolapse in the onset and progression of regionalized left ventricular fibrosis. Our findings further support modifying surgical guidelines to allow for earlier valve repair, in order to decrease LV fibrosis and minimize postoperative LV dysfunction in MVP patients.

Session 2: Neuroscience

Lincoln Wurtz

Mayo Clinic

The Dynamic Role of Kallikrein 6 in Myelination

Kallikrein related peptidase 6 (Klk6) is a secreted serine protease highly expressed in oligodendrocytes and implicated in demyelinating conditions. To gain insights into the significance of Klk6 to oligodendrocyte biology, we investigated the impact of global Klk6 gene knockout on CNS developmental myelination using the spinal cord of male and female mice as a model. Results demonstrate that constitutive loss of Klk6 expression accelerates oligodendrocyte differentiation developmentally, including increases in the expression of myelin proteins such as MBP, PLP and CNPase, in the number of CC-1+ mature oligodendrocytes, and myelin thickness by the end of the first postnatal week. Co-ordinate elevations in the pro-myelinating signaling pathways ERK and AKT, expression of fatty acid 2-hydroxylase, and myelin regulatory transcription factor were also observed in the spinal cord of 7d Klk6 knockouts. LC/MS/MS quantification of spinal cord lipids showed sphingosine and sphingomyelins to be elevated in Klk6 knockouts at the peak of myelination. Oligodendrocyte progenitor cells (OPCs)-derived from Klk6 knockouts, or wild type OPCs-treated with a Klk6 inhibitor (DFKZ-251), also showed increased MBP and PLP. Moreover, inhibition of Klk6 in OPC cultures enhanced brain derived neurotrophic factor-driven differentiation. Altogether, these findings suggest that oligodendrocyte-derived Klk6 may operate as an autocrine or paracrine rheostat, or brake, on pro-myelinating signaling serving to regulate myelin homeostasis developmentally and in the adult. These findings document for the first time that inhibition of Klk6 globally, or specifically in oligodendrocyte progenitors, is a strategy to increase early stages of oligodendrocyte differentiation and myelin production in the CNS. Beyond developmental myelination, this study warrants future research into the therapeutic potential of blocking Klk6 to promote myelin regeneration in demyelinating conditions such as multiple sclerosis.

Sarah Zych

University of Colorado

Divergent properties and independent regulation of striatal dopamine and GABA co-transmission

Neuronal populations that release multiple neurotransmitters enable a dynamic range of signaling through the spatial and temporal scales of co-transmitter release. Within the basal ganglia, midbrain dopamine (DA) neurons co-release GABA from axon terminals in the striatum, facilitating learning, movement, motivation, and reward. GABA co-release plays a role in addiction as reduced co-release leads to increased ethanol intake and preference in mice. However, little is known about the mechanism or functional release properties of co-released GABA or how it shapes DA signaling. As DA and GABA are both loaded into synaptic vesicles by the vesicular monoamine transporter 2 (VMAT2), it follows that they should be released together with the same properties. To investigate the properties of DA and GABA co-transmission, we used whole-cell voltage-clamp recordings of D2- and GABAA-receptor mediated inhibitory post-synaptic currents (IPSCs) activated by DA and GABA co-release onto striatal medium spiny neurons (MSNs). Surprisingly, we found that DA and GABA exhibit different release properties. We found that DA release is more sensitive to calcium sources and exhibits looser coupling between calcium entry and calcium sensing release machinery. DA and GABA release is also differently modulated by presynaptic GABAB, kappa opioid, and D2 receptors which more strongly modulate the release of DA than GABA. In addition, we found that DA and GABA have different release probabilities and rely on different active zone scaffolding proteins. Genetic removal of the scaffolding protein RIM abolished the high release probability of DA, but GABA release was unaffected. Surprisingly, despite DA and GABA being packaged by VMAT2, our data indicates that intrinsic differences exist between transmitter release, endowing DA neurons with the ability to independently regulate co-transmitted signals and thus fine-tune the spatiotemporal scale of dopaminergic signaling. We theorize that DA and GABA either occupy separate vesicle populations or are co-packaged and spatially segregated into different active zones opposing D2 or GABAA receptors. Understanding the mechanism and balance of DA/GABA co-transmission elucidates the complexity of dopaminergic signaling and opens the door to study how these dynamics shift in disease states.

Tyrone DeSpenza

Yale School of Medicine

Therapeutic targeting of mTOR in a genetic subtype of congenital hydrocephalus

Congenital hydrocephalus (CH), featuring enlarged brain ventricles, has been classically attributed to failed cerebrospinal fluid (CSF) homeostasis and treated with invasive surgical procedures with high morbidity and failure rates. Significant gaps in our understanding of the molecular mechanisms of human CH impede the development of preventive measures and targeted therapies. A personalized medical approach presents opportunities to enhance therapeutics for CH patients beyond surgical interventions, leveraging molecular genomics to identify pathogenic mechanisms that may be exploited pharmacologically. Using whole exome sequencing (WES), we recently demonstrated de novo mutations (DNMs) in four different neural stem cell (NSC) regulator genes cause >10% of sporadic, CSF-shunted human CH (Neuron, 2018). All four genes regulate NSC fate, implicating dysregulated NSC development in a subset of CH patients. In subsequent WES studies >250 CH patients, we have identified the PTEN-PI3K-mTOR signaling pathway as the most commonly mutated gene module in sporadic human CH to date, with multiple loss-of-function DNMs in PTEN (Nat Med, 2020). Corroborating our human genetic findings, we have found that embryonic conditional deletion of Pten in a discrete subset of NSCs is sufficient to cause early postnatal lethality and CH in mice. We use postnatal delivery of everolimus, an FDA-approved mTOR inhibitor, and concomitant conditional genetic deletion of Raptor, a critical component of mTORC1, to promote survival and attenuate the progression of CH in Pten cKO mice. We aim to

provide insight into the cellular and molecular mechanisms of an NSC-driven, genetic subtype of human CH, setting the stage for near-term pre-clinical and translational studies for individuals diagnosed with CH and harboring PTEN mutations.

Akansha Jain

University of Iowa

Modulation of cellular ATP levels as a potential therapeutic strategy for Parkinson's disease

Parkinson's disease (PD) is a neurodegenerative disorder affecting approximately one million Americans. Treatments for PD fail to prevent or slow neuron loss. Three factors suggest that increasing cellular ATP levels might serve as a therapeutic strategy for PD. First, aging, environmental toxins, and genetic mutations are all risk factors for PD, and all impair energy metabolism and decrease cellular ATP levels. Second, a pathological hallmark of PD is the presence of Lewy bodies that contain alpha-Synuclein (αSyn) aggregates, and physiologic concentrations of ATP, by acting as a hydrotrope, can prevent formation of and dissolve previously formed protein aggregates. Third, increasing brain ATP levels with a pharmacologic agent, terazosin (TZ), is neuroprotective in toxin-induced and genetic models of PD in mice, rats, flies, and induced pluripotent stem cells. TZ, an FDA-approved drug, binds to and enhances the activity of the ATP-generating glycolytic enzyme, phosphoglycerate kinase 1 (PGK1). The goal of this study was to test specific interventions that modulate glycolysis and further understand the mechanism of TZ. To increase cellular ATP levels, we targeted PGK1. Pharmacological activation of PGK1 with TZ increased cellular ATP levels. Knockdown of PGK1 decreased ATP levels and attenuated the effect of TZ. Conversely, overexpressing PGK1 increased ATP levels and mirrored the effect seen with TZ. To assess metabolic pathways affected by TZ, we metabolically profiled HEK293T cells and found that TZ increased metabolites of two main pathways: the TCA cycle and the pentose phosphate pathway. To understand functional changes with TZ and PGK1 expression levels, we assessed cellular bioenergetics with seahorse assays. TZ increased glycolytic function and the increase depended on PGK1. Consistent with this, PGK1 overexpression also increased glycolytic function, and PGK1 knockdown decreased glycolytic function. Collectively, these data suggest that increasing PGK1 activity increased glycolytic function and the TCA cycle and thereby increased cellular ATP levels. Lastly, PGK1 overexpression in HeLa cells expressing the A53T mutant form of αSyn decreased aggregation. In future experiments, I will study the mechanism by which stimulating glycolysis affects αSyn liquid-liquid phase separation.

Session 2: Molecular & Cellular Biology

Andrea Toth

University of Cincinnati

Understanding and preventing perinatal lung injury in a rhesus macaque model of chorioamnionitis

Introduction: Damage to the developing human lung causes perinatal morbidity and mortality with lifelong consequences on pulmonary health. Chorioamnionitis impacts 25-40% of preterm births and is associated with higher risk of pulmonary disease in childhood. Understanding perinatal human lung injury is challenging due to limited availability of human tissue and because other common model systems do not accurately model human pulmonary development. Human lungs require a functional gas exchange surface composed of mature pulmonary alveoli at birth, containing distinct cellular and regional organization. We therefore characterized distal lung

development in an LPS-induced model of chorioamnionitis in prenatal rhesus macaque (Macaca mulatta), a model sharing these critical aspects of human lung development, to test the impact of inflammation on fetal lung.

Results: Using high content imaging and scRNAseq, we developed an atlas of primate lung development, identifying signaling patterns and cellular relationships during alveologenesis. Chorioamnionitis caused significant inflammatory injury to the lung, disrupting the complex multicellular alveolar signaling niche, leading to alveolar simplification, disruption/loss of gas exchange surface, and pathology similar to that found in chronic lung disease of newborns. Blockade of key chorioamnionitis-associated cytokines IL-1b and TNFa ameliorated inflammatory lung injury by modulating innate immune activation in myeloid cells and blunting stromal response to inflammation, directly implicating these cytokines in the mechanisms of lung injury during chorioamnionitis. Conclusions: These data refine our understanding of the pathophysiology of chorioamnionitis-mediated lung injury via direct evidence that perinatal inflammation in chorioamnionitis causes injury to the developing primate lung. Inflammatory blockade prevented severe lung injury, suggesting that targeted immunomodulatory therapies may treat human chorioamnionitis.

Kelsey Nolden

Medical College of Wisconsin

A novel Fis1 inhibitor reverses diabetic endothelial dysfunction in human resistance arteries

Microvascular dysfunction contributes to multiple diseases, including diabetes mellitus, and has been associated with mitochondrial dysfunction. We have found that endothelial cells from diabetic patients have hyper-fragmented mitochondrial networks, increased Fis1 expression, increased reactive oxygen species (ROS), and impaired function associated with increased cardiovascular risk. Mitochondrial fragmentation Medical College of Wisconsin appears to derive from excessive fission as knockdown of fission proteins in cultured endothelial cells restores mitochondrial morphology and nitric oxide (NO) bioavailability. The role of Fis1 in endothelial function was investigated using overexpression and genetic silencing of Fis1 in ex vivo patient-derived small resistance arteries and HMEC1 cells. Gene and protein expression profiles, mitochondrial respiration, and electrical resistance measurements support the hypothesis that Fis1 is essential for maintaining normal endothelial cell layer barrier function. Molecular suppression of Fis1 reverses impaired endothelium dependent vasodilation in vessels from humans with type 2 diabetes, as well as healthy human vessels exposed to high (33 mM) or low (2.5 mM) glucose concentrations (N=6 per condition, P<0.001). Conversely, overexpression of Fis1 in healthy vessels induces impaired vasodilation (N=5, P<0.001), suggesting a causative role. A novel Fis1 inhibitor, pep213, was developed and found to bind human Fis1 with a low micromolar affinity (3.3-7 μM). A 1.85 Å co-complex crystallography structure of Fis1-pep213 confirms inhibitor specificity. Application of pep213 reverses impaired endothelium-dependent vasodilation of vessels from patients with type 2 diabetes (N=5, P<0.001), as well as healthy controls exposed to high glucose concentrations (N=5, P<0.001), by improving NO bioavailability and decreasing excess ROS generation. These data support a potentially novel therapeutic route for treating diabetic microvascular disease through Fis1 inhibition.

Juan Colazo

Vanderbilt University

Albumin-hitchhiking MMP13 siRNA (siMMP13<(EG18L)2) for the treatment of Rheumatic Disease

Background: Osteoarthritis (OA) and rheumatoid arthritis (RA) decrease quality of life due to joint destruction, pain, and decreased function. Multiple joint osteoarthritis (MJOA) can occur in up to

50% of OA cases. The high incidence of MJOA motivates the development of systemic therapies. Although there are successful therapies for RA, many patients do not benefit, and they can cause side effects. In OA/RA, matrix metalloproteinases (MMPs) drive joint degeneration. Small molecule MMP inhibitors were tested clinically, but caused toxicities due to lack of MMP selectivity. Short-interfering RNA (siRNA) can be used to silence “undruggable targets” but are limited due to significant biological barriers. Albumin-based drug delivery shows promise due to its high half-life, recycling ability, and accumulation in diseased joints. Herein, we characterize a chemically stabilized MMP13 siRNA molecule, siMMP13<(EG18L)2, which spontaneously binds albumin 'in situ'.

Methods: The siRNA was synthesized with alternating 2′-OMe and 2′-F ribosugar modifications to stabilize against endonucleases and phosphorothiate linkages on the backbone to block exonucleases. For albumin-binding, a splitter phosphoramidite (<) (to allow for diacyl addition i.e., 2 lipid tails) at the 5' end of the sense strand was added followed by 3 repeats of a hexa-ethylene glycol (EG6) phosphoramidite. Thus, 18 EGs were added prior to each eighteen-carbon (C18) acyl (2x, L2 nomenclature). An overload-induced mouse model (9N, 250 cycles, 3x/week) and the K/BxN serum transfer arthritis mouse model were used for OA and RA studies, respectively.

Results: RNA stabilization chemistries increased serum/synovial fluid stability, while maintaining potent MMP13 silencing. Albumin-binding dye Evan’s Blue and fluorescent albumin accumulated more in OA knees/K/BxN hindpaws than healthy knees/hindpaws. siMMP13<(EG18L)2 showed preferential delivery to OA knees/K/BxN hindpaws over healthy knees/hindpaws and demonstrated greater pharmacokinetics than non-end-modified siRNA and cholesterol-conjugated siRNA. In the K/BxN model, siMMP13<(EG18L)2 reached/treated multiple joints including forepaws, knees, and hindpaws. Overall, siMMP13<(EG18L)2 treatment was safe, provided potent MMP13 mRNA/protein knockdown, provided joint pain benefits, reduced arthritis clinical score, reduced arthritis-related genes, provided joint protection, and performed better than or equal to gold standard clinical controls.

Conclusions: Albumin-hitchhiking siRNA shows promise as a platform technology that can be readily adapted for targeting of many rheumatic disease-driver genes, and potentially used in other diseases. Furthermore, this siRNA technology is modular allowing for engineering of many other chemistries such as targeting/tethering moieties, etc.

Javier Sierra Pagan

University of Minnesota

ETV2 functions as a pioneer transcription factor that regulates endothelial lineage development and reprogramming

The vasculature is an essential organ for the delivery of blood and oxygen to all tissues of the body and is therefore relevant to the treatment of ischemic heart disease. The ETS-related transcription factor 2 (ETV2) is a transcription factor that is both necessary and sufficient for the development of hemathoendothelial lineages. The molecular mechanism by which ETV2 promotes endothelial development is not known and warrants further investigation. Here, we report that ETV2 functions as a novel pioneer transcription factor that relaxes heterochromatin to regulate endothelial development. Pioneer factors are a subset of transcription factors that bind heterochromatin and promote chromatin relaxation in order to drive the development and reprogramming of different cell lineages. By comparing engineered embryonic stem cell differentiation and fibroblast reprogramming models capable of overexpressing ETV2 with single cell RNA-seq, ATAC-seq, ChIP-seq and in-vitro nucleosomal binding techniques, we demonstrated that ETV2 was able to bind nucleosomal DNA or heterochromatin to function as a pioneer factor independent of the cellular context. We also determined that ETV2 executed a pioneering role by recruiting and directly interacting with the ATP-dependent chromatin remodeling enzyme BRG1 to remodel chromatin around endothelial genes to help maintain an open configuration, resulting in increased H3K27ac

deposition. Additionally, the ETV2-BRG1 complex functioned to recruit ELK3 to further drive endothelial lineage development. Collectively, these results that define a novel pioneer factor and epigenetic regulatory mechanisms that govern the specification and differentiation of endothelial progenitor cells will serve as a platform for targeted therapies to promote cardiovascular regeneration by generating new vasculature.

Session 3: General Medicine I

Havell Markus

Pennsylvania State College of Medicine

Utilizing electronic health records to identify high risk ANA-positive patients that may progress to systemic lupus erythematosus

Systemic lupus erythematosus (SLE) is a severe and heterogeneous autoimmune disease. SLE is often preceded by a stage with milder symptoms and positive antinuclear antibody (ANA+) tests, which we call Incomplete Lupus Erythematosus (ILE). Only 10-50% ILE patients progress to SLE. Deriving biomarkers that can forecast ILE to SLE progression is important for early diagnosis and interventions.

We utilized electronic health record (EHR) data from the TriNetX Research Network to derive biomarkers that can forecast disease progressions from ILE to SLE. ANA+ patients were identified using LOINC laboratory test definitions. An ANA+ individual is deemed progressing (or not progressing) to SLE, if they have (or do not have) SLE diagnosis within 6 months to 3 years after first ANA+ test. A 6-months period between ANA+ and SLE diagnosis is set to make sure that ANA+ individuals would not have had SLE at the time of the positive tests. To minimize the impact of censored data, we only included patients who had clinical data available at least 1 year before and 3 years after the ANA+ diagnosis. We utilized this data to train machine learning models to predict SLE diagnosis after ANA positive test result. Training and testing of these models were carried out using patients in the Research Network (44,031 controls and 1,309 cases) and Diamond Network (27,274 controls and 1,016 cases) subset of TriNetX respectively.

Simple linear regression model of individual clinical predictors (controlling for age, sex, and race) identified marginally significant EHR codes involving the immune system, peripheral opioid receptor antagonists, and basophil number as significant predictors of future SLE diagnosis. Using the marginally significant predictors, we train a gradient boosting model (GBM), which yielded an area under curve for the receiver operation curve of 0.64 in testing dataset. Additonally, the predicted value from the GBM model can stratify ANA+ patients that have high risk of progressing to SLE. Specifically, patients with GBM predicted values in the top 5th percentile have more than 3x increased risk of progression (9%) from ILE to SLE compared to individuals with predicted values in the bottom 5th percentile (3%).

EHR could be utilized for identifying high risk ANA positive patients likely to progress from ILE to SLE. Given that EHR data does not incur additional cost of data collection, it could be readily used in clinic and assist clinicians to screen patients with high risk of progressing from ILE to SLE.

Conclusions: Albumin-hitchhiking siRNA shows promise as a platform technology that can be readily adapted for targeting of many rheumatic disease-driver genes, and potentially used in other diseases. Furthermore, this siRNA technology is modular allowing for engineering of many other chemistries such as targeting/tethering moieties, etc.

Simone Herzberg

Vanderbilt University

Obesity and Rotator Cuff Disease: A Systematic Review and Meta-Analysis

Background: Rates of obesity continue to rise, with nearly 50% of the US population classified as obese. Several studies have investigated the relationship between obesity and rotator cuff disease (RCD), but results are not consistent.

Purpose: Obesity is a modifiable risk factor and understanding of its impact on RCD will inform better patient care and potentially mitigate incidence and progression of RCD. Objective: Synthesize evidence on the association of obesity and RCD from peer-reviewed published literature. Study Design: Systemic Review/Meta-Analysis.

Methods: We employed search terms to execute systematic queries of PubMed, Embase, Cochrane, CINAHL, and Science Direct up to Dec 8, 2020. Articles meeting eligibility criteria and reporting on the association of obesity and RCD were considered. Meta-analysis was performed to quantitatively summarize associations between the most common measure of obesity, body mass index (BMI), and RCD to report odds ratios (OR), and corresponding 95% confidence intervals (CI) for regression-based models and BMI mean differences (MD) between cases and controls. Studies reporting ORs for BMI as a continuous measure, and as categorical measures [normal weight, overweight, and obese] were recorded and aggregated separately. We performed a risk of bias (RoB) assessment of studies with the ROBBINS-I tool.

Results: After a full-text review of 218 articles, 12 articles assessing measures of obesity were eligible for meta-analysis. Individuals with RCD were 1.16 times as likely to be overweight (OR: 1.16; 95% CI: 1.05, 1.29), and 1.29 times as likely to be obese (OR = 1.29; 95% CI: 1.16, 1.44) as compared with individuals without RCD. Each unit increase in BMI was associated with 3% higher odds of having RCD (OR = 1.03; 95% CI: 1.02, 1.04). On average, individuals with RCD had 0.48 kg/m2 higher BMI than individuals without (MD: 0.48 95% CI: 0.11,0.84). RoB assessment of all studies eligible in systematic review suggested confounding and inability to establish temporality as major issues in existing studies.

Discussion: Being overweight or obese was associated with significantly higher odds of RCD. However, most studies on this topic are not optimal in design and analysis. Targeting obesity as a modifiable risk factor for RCD could help decrease the prevalence or progression of RCD and improve patient care.

Al Smith

Carle Illinois College of Medicine

Quantifying the Need for Global Emergency Neurosurgical Intervention

Introduction: EVD placement is one of the mainstay therapies for the management of elevated intracranial pressure. Indications include traumatic brain injury, hydrocephalus, meningitis, and various other pathologies. While there is a marked paucity of neurosurgical care globally, the number of patients who require an EVD are frequently found in low-middle income areas. For this reason, we seek to quantify the need for EVD placement globally to determine regions that would benefit from technological advancement in the domain.

Methods: Worldwide distribution of neurosurgeons with the incidence of indication for EVD (TBI, meningitis, and hydrocephalus) were compiled from the World Neurosurgery Workforce in 20161-3. The aggregated values were scaled from 0 to 1 relative to their minimum and maximum. A model was constructed with neurosurgeon distribution comprising 60% of the model, and indications for EVD comprising 40% of the model. Indication scores were computed using this model and visually represented using the rworldmap package in R.

Results: Our global analysis found Montenegro, with an indication score of 0.776, Equatorial Guinea, with an indication score of 0.709, and the Bahamas with an indication score of 0.708 to be the top three targets for improved access to neurosurgical care based on the low numbers of

practicing neurosurgeons and high indications for neurosurgical treatment. The generated world map shows the comparative analysis of neurosurgical need for different countries, with darker regions indicating a higher need for neurosurgical access due to more indications for treatment.

Conclusions: Emergencies require neurosurgical access within hours of the inciting event, resulting in major burden to regions with high incidence of neurosurgical emergencies such as severe TBI. Our analysis shows that targeting select regions for deployment of neurosurgical resources and development of new neurosurgical technology for guided EVD placement may reduce the global burden of emergency neurological conditions.

Lily Nguyen

University of Colorado

Combination epigenetic and PARP inhibitor therapies can uncover transposable elements with gene regulatory function in ovarian cancer cells

Introduction: Transposable elements (TEs), which make up nearly 50% of the human genome, are a rich source of gene regulatory motifs. However, their gene regulatory functions are rarely tested. Here, we used a combination of an epigenetic therapy, EHMT1/2 inhibitors (EHMTi), and an FDA-approved maintenance therapy for ovarian cancer, PARP inhibitors (PARPi), to uncover TEs with gene regulatory motifs and test their functions in ovarian cancer cells.

Methods: Several ovarian cancer cell lines were treated with DMSO or combination (combo) therapy of an EHMTi (UNC0642) and PARPi (Olaparib). RNA-sequencing and CUT&TAG were used to determine if this combo therapy upregulates TEs on the transcript and epigenome level, respectively. Bioinformatic pipelines DESeq2, MACS2, TEtranscripts, and Giggle were used to analyze our transcriptomic and epigenomic data. CRISPRi was used to silence specific TE and gene loci.

Results: Combo therapy was successful in reactivating transposable elements on the transcript and epigenomic level. On the transcript level, several TE families have increased transcription upon treatment. Combo therapy also resulted in a global upregulation of gene expression. On the epigenome level, the same TE families with increased transcript levels also have increased enhancer marks. Combining both the transcriptomic and epigenomic data, we found putative pairs of combo-upregulated TE-derived enhancers and genes. Of interest is the tumor suppressor gene, TIPARP, and the TE-derived enhancer, LTR18, located ~47Kbp upstream of TIPARP. Excitingly, we found that silencing LTR18 alone is sufficient to decrease TIPARP expression, suggesting that TEs do indeed have gene regulatory functions. We also reanalyzed publicly available datasets of normal and tumor ovarian cells and saw that LTR18 enhancer activity is globally decreased in tumor cells compared to normal cells. This suggests that TE-derived LTR18 enhancer activity is lost as normal ovarian cells transform into tumor cells and that this loss of enhancer activity has unexpected effects on gene expression.

Conclusion: Our studies identified a novel TE-derived enhancer, LTR18, that regulates the expression of a tumor suppressor gene, TIPARP. These studies will give insights to non-canonical ways in which cells can regulate gene expression.

Session 3: General Medicine II

Jared Beyersdorf

Emory University / Georgia Institute of Technology

Robust, Durable Gene Activation In Vivo via mRNA-Encoded Activators

Programmable control of gene expression via dCas9 fusion proteins has enabled the engineering of cellular behaviors. Viral vectors have demonstrated the functionality of these approaches in vivo, but questions remain regarding the safety of AAV and other virus-based approaches. There is a need for safer delivery strategies for large dCas9 fusion proteins. Given the recent successes of synthetic mRNA for vaccines and their inherently improved safety profile, we developed synthetic mRNAs expressing dCas9 fusion proteins and hypothesized that lipid nanoparticles (LNPs) would facilitate mRNA and sgRNA delivery to enable efficient gene activation in vivo. As an initial model gene, we chose the glycosyltransferase B4galnt2 as it is not highly expressed in the liver of mice, and the effect of its protein function can easily by detected via lectin-based staining. We delivered optimized LNPs containing synthetic mRNAs and targeted sgRNAs to wild-type mice by tail vein. We achieved a 10^6-fold increase in transcription from the B4galnt2 gene in ~99% of all liver hepatocytes. Furthermore, we demonstrated that a single injection of LNPs produced sustained activation of the B4galnt2 gene for over 10 days with the ability to re-dose. We then showed that gene activation could be inhibited or blocked using mRNAs encoding anti-CRISPR proteins, which further improve the safety profile of this approach. Finally, we used this technology to upregulate the erythropoietin gene, which encodes a protein hormone that promotes erythropoiesis. Seven days after a single injection of an LNP therapeutic targeting the erythropoietin gene, we observed a significant increase in hematocrit of treated mice from ~50% to ~63%. Importantly, this approach does not edit target genes and can be applied in wild type animals. Overall, our results demonstrate that a synthetic mRNA-based approach can be used for safe and programmable control of gene expression in vivo.

Mary Adeyeye

University of Texas

Paradoxical Effect of Frizzled2-Fc on Bone Mass for the Treatment of Osteogenesis Imperfecta

Osteogenesis imperfecta (OI) is a group of genetically and phenotypically heterogeneous connective tissue disorders that results in low bone mass, bone deformity, and bone fractures. OI has an estimated prevalence of 1 in 15,000 births. Disruptions in multiple processes such as collagen synthesis, collagen posttranslational modification, signaling defects and intracellular trafficking lead to OI. The primary focus of medical therapy has been to increase bone mass and reduce fracture risk through medical and surgical treatment. The mainstay of treatment in this population is bisphosphonates, which reduces bone loss by suppressing bone turnover. However, these drugs can only delay bone loss without fully preventing it. Wnt signaling is an anabolic pathway in bone and activating the Wnt signaling pathways have been shown to increase bone formation. Surprisingly, very little is known about frizzled receptors, which are one of the main co-receptors that mediate the anabolic effects by the Wnt signaling pathway. In preliminary studies, we have shown Frizzled2-Fc (Fz2-Fc) paradoxically increases bone mass in wild-type mice after a single dose of treatment, one week later. We tested whether this reagent can be used therapeutically in the context of OI. We studied the potential application for treatment of both collagen and Wnt1-related forms of OI and investigate the mechanism by which Fz2-Fc increase in bone mass. We treated OI mouse models (Col1a2tm1.Mcbr, Crtap-/-, and Dmp1-Cre;Wnt1fl/fl) with Fz2-Fc signaling. Mice were treated at 3 months old with Fz2-Fc weekly for 8 weeks. Bone tissues for microCT analysis, bone histomorphometry, biomechanical testing, histology, and RNA and protein analysis. Our studies indicate that repeated treatment with Fz2-Fc increases bone mass in not only wild type (WT) but also dominant (Col1a2tm1.Mcbr) and recessive models of OI (Crtap-/- and Dmp1-Cre;Wnt1fl/fl). Mice treated long term with Fz2-Fc had significant increases in bone mass, bone surface and bone volume. This paradoxical effect could be translated into therapy and raises the questions about potential components of the Wnt signaling pathway, that could be augmenting bone formation.

Maryknoll Linscott

Pennsylvania State College of Medicine

PIK3CA and cMYC promote the expansion of distinct Ras-initiated, long-lived premalignant clones in a multistage murine breast cancer model

A remote carcinogen exposure can predispose to a clinical presentation of breast cancer decades later. Standard multi-stage carcinogenesis models posit that carcinogen-induced mutations generate long-lived premalignant clones, which acquire additional oncogenic mutations as they evolve toward invasive cancers. However, the biological features of the earliest clones “initiated” on the path to cancer by carcinogen-induced mutations remain obscure, hindering the rational design of chemoprevention strategies. In prior work, we showed that a one-time exposure to carcinogen 7,12-dimethylbenzanthracene (DMBA) generates initiated mammary epithelial cell (iMEC) clones bearing signature *Hras*^{Q61L} mutations, which remain subclinical indefinitely until inducible activation of oncogenic Wnt signaling triggers their rapid clonal expansion into malignant outgrowths. Here, we adapted this multistage model by replacing Wnt pathway activation with inducible expression of either *c-MYC* or *PIK3CA*^{H1047R}, recapitulating two of the most prevalent oncogenic events in human breast cancer. Despite using a DMBA exposure identical to that used in our Wnt work, neither inducible *c-MYC* (iMYC) expression nor inducible *PIK3CA*^{H1047R} (iPIK) expression efficiently selected for outgrowth of *Hras*^{Q61L} iMEC clones. Instead, iMYC and iPIK expression selected for outgrowth of iMECs bearing activating mutations in distinct Ras family genes, with iMYC expression promoting *Kras*^{mut} and *Nras*^{mut} tumors and iPIK expression prompting the development of *Kras*^{mut} tumors. Selection for these preferred Ras family mutations occurred whether oncogene expression was induced within days of DMBA exposure or months later. Thus, DMBA exposure generated a wide variety of long-lived, clinically silent iMEC clones bearing different Ras family driver mutations in iPIK and iMYC mice. Similar to our Wnt work, early parity (full-term pregnancy) failed to eliminate established iMECs in iPIK and iMYC mice. However, our parity-induced protection schemes shifted the spectrum of *Ras*^{mut} iMEC clones in iPIK and iMYC mice. Whereas, iMYC expression promoted the expansion *Nras*^{mut} iMEC clones, both iPIK and iMYC expression selected against outgrowth of *Kras*^{mut} clones, suggesting that the latter iMECs may be sensitive to parity protection. Together, our findings demonstrate that oncogenes *PIK3CA* and *cMYC* select for the expansion of long-lived, premalignant clones carrying distinct *Ras* mutations and sensitivity to parity protection. Further investigation of the cellular and molecular mechanisms underlying the differential selection of these premalignant clones may uncover targets for breast cancer chemoprevention.

Kyle Woisard

Virginia Commonwealth University

Executive Control Network Resting State fMRI Functional Connectivity in Cocaine Dependent Subjects

Resting state functional magnetic resonance imaging (fMRI) functional connectivity has been used as a tool to study brain mechanisms associated with addictions. However, most studies have focused on the default mode network (DMN) with fewer studies assessing the executive control network (ECN), despite well-documented executive control deficits in addictions. The present study assessed the functional connectivity within and between the ECN, DMN, and salience network (SN) in cocaine dependent subjects (CD) compared to healthy control subjects (HC) using independent component analysis, dual regression, and FSLNets. This study also examined the relationship between functional connectivity and delayed discounting scores. In CD (n=22) compared to HC (n=22), a cluster in the right precentral and postcentral gyri within the right ECN had trend stronger functional connectivity. No significant differences were found between groups for the functional

connectivity of the DMN, SN, or right ECN, or between any of the networks examined. No significant associations between functional connectivity and delayed discounting scores were found. These novel preliminary findings provide tentative support that ECN functional connectivity may differ between CD and HC. These results, if verified with a larger sample size, would be consistent with previous research showing differences in ECN functional connectivity in addictions and altered executive function in CD.

Session 4: Immunology

Jacqueline Turner

University of Colorado

Lysophosphatidic acid rewires CD8 T cell metabolism and anti-tumor immunity

Lysophosphatidic acid (LPA) is bioactive lipid mediator which is elevated both locally and systemically across different cancer types. It remains unknown if and/or how LPA modifies metabolic demands, cell stress, and differentiation programs in cytotoxic CD8 T cells to regulate adaptive immunity. We hypothesized signaling through LPAR promotes tolerogenic states through metabolic reprogramming and potentiates exhaustive differentiation to modulate anti-tumor immunity. Using metabolic studies, we determined LPA rewires metabolism and modulates reactive oxygen species accumulation. Specifically, we show LPA signaling through LPA receptor 5 (LPAR5) regulates CD8 T cell respiration, proton leak, and reactive oxygen species. We demonstrate metabolic efficiency/performance and antigen specific CD8 T cell killing is modulated by LPAR5. Further, adoptive transfer of LPAR5 receptor knockout CD8 T cells into tumor bearing mice expressed fewer markers of exhaustion and had significantly improved anti-tumor immunity. To further investigate the role of LPA in cancer, we performed lipidomics on plasma samples from stage IV melanoma patients and measured LPA pre- and post-immunotherapy treatment. We found lower LPA levels predicted response to immunotherapy. In summary, we show LPA modulates CD8 T cell metabolic programming, CD8 T cell function, and exhaustive differentiation. Our study offers key insights into the mechanisms governing adaptive anti-tumor immunity and identify metabolic inhibition targeting lipid signaling as a novel approach to prevent CD8 T cell dysfunction and or reinvigorate dysfunctional CD8 T cells.

Peter Dimitrion

Wayne State University

Dissecting peripheral and cutaneous immunological dysregulation in patients with hidradenitis suppurativa

Hidradenitis suppurativa (HS) is a chronic inflammatory skin disease with multifactorial etiology that harbors a complex cutaneous immune reaction localized primarily affecting intertriginous skin. High rates of systemic inflammatory comorbidities and dysregulation of the serum proteome point to systemic inflammation as a hallmark of disease. Thus, we hypothesized that patients with HS harbored markedly dysregulated peripheral immunomes. We analyzed fresh whole blood samples (18 moderate-to-severe HS and 12 healthy controls (HC)) analyzed by mass cytometry (CyTOF) using a 30-marker panel to determine peripheral immune profiles. Peripheral immunomes of patients with HS exhibited decreased natural killer cells, dendritic cells, classical and nonclassical monocytes; and increased Th17 cells and intermediate monocytes (IMs). Cluster identification, characterization, and regression analysis confirmed these findings and further revealed a subpopulation of IMs with elevated expression of CD38 and skin-homing chemokine receptors (shCCRs). All monocyte subsets increased shCCRs in a CD38 dependent manner in patients with

HS. CD38 metabolizes NAD+, which has numerous downstream effects, including elaboration of inflammatory mediators and chemotaxis by myeloid cells. CD38+ IMs were the only cell type elevated in moderate HS. We performed a meta-analysis of all publicly available bulk RNA-seq data from HS skin and found increased CD38 expression in lesional HS skin, but not perilesional skin. Genes correlated with CD38 expression ($r^2>0.5$; $p<0.05$) enriched for an autoinflammatory gene signature and immune infiltration from the blood. We confirmed this by immunohistochemistry (20 HS lesions and 6 perilesions). Staining patterns suggested that cells other than immune cells upregulate CD38 in lesional HS skin. Using imaging CyTOF we assayed cellular neighborhoods in lesional and perilesional skin and found patches of infiltrating CD38+ monocytes and CD38+ endothelial cells in proximity and increased vascularization in lesional HS skin. Together these results highlight the potential for CD38+ IMs as an early cellular biomarker and identifies a putative monocyte-endothelial communication axis promoting immune cell infiltration and recruitment in HS lesions.

Sara Bolivar Wagers

University of Minnesota

Prevention of acute graft-versus-host disease using an orthogonal IL-2/IL-2Rβ system to selectively expand regulatory T cells in vivo

Allogeneic hematopoietic stem cell transplantation (allo-HSCT) is a curative option for many hematological disorders. However, Graft versus Host Disease (GVHD) remains a leading complication after transplant. Regulatory T cell (Treg) therapies have proven efficacious in ameliorating GVHD but are limited by variable suppressive capacities and need for a high therapeutic dose. In this study, we sought to expand Treg in vivo by engineering murine Treg to express an orthogonal IL-2Rβ (oRβ) that would selectively interact with its orthogonal IL-2 (oIL-2) cytokine and would not interact with its wildtype counterpart. To test whether the orthogonal system would preferentially drive donor Treg expansion, we used a murine MHC-disparate GVHD model of lethally irradiated BALB/c mice given T-cell depleted bone marrow from C57BL/6 (B6) mice alone, or together with B6-GFP control or oRβ transduced Treg. On day 2, B6 Tcons were injected to induce GVHD. Recipient mice were treated with PBS or oIL-2 daily for 14 days, then 3 times weekly for 14 days. Mice treated with oRβ Treg and oIL-2 vs PBS had enhanced GVHD survival, in vivo selective expansion of Treg and a greater decrease in activated Tcons in peripheral blood, mesenteric lymph node, spleen, and intestines. Importantly, oRβ Treg maintained graft versus leukemia (GvL) responses in two distinct tumor models (A20, MLL-AF9). Thus, these data support the application of a novel approach to enhance the efficacy of Treg cell therapy in allo-HSCT setting using an oIL-2/oRβ system that allows for selective in vivo expansion of Treg leading to GVHD protection and GVL maintenance.

Jonathan Liang

Yale School of Medicine

Saturated fatty acid activation of the NLRP3 inflammasome depends more strongly on JNK activation than on ROS

Diseases associated with obesity, such as atherosclerosis and fatty liver disease, involve chronic inflammation of fat-exposed organs. One mechanism that contributes to inflammation in these diseases is the NLRP3 inflammasome, a macromolecular complex that responds to diverse danger signals by inducing secretion of inflammatory IL-1 family cytokines and promoting a type of cell death called pyroptosis. Saturated fatty acids (SFAs) are known to activate the NLRP3 inflammasome but the intracellular pathways by which this activation occurs are incompletely described. We investigated the signaling mechanisms required for NLRP3 activation by the most biologically-abundant SFA, palmitic acid (PA), through a combination of imaging and pathway

inhibition. We report that NLRP3 activation by PA, unlike activation by classical NLRP3 triggers such as nigericin, is only weakly dependent on reactive oxygen species (ROS). While classical activators engage multiple ROS sources including both mitochondrial and cytosolic enzymes, PA activation of NLRP3 specifically requires ROS from NADPH oxidases. PA and nigericin both require the MAP kinase JNK to activation NLRP3, but the details of JNK activation differ significantly between the two triggers. While nigericin activates mostly nuclear JNK, PA leads to cytosolic JNK activation that does not depend on the upstream MAP3 kinases TAK1 and ASK1 that are important for nigericin. In addition, we investigate the unsaturated fatty acid arachidonic acid (AA), demonstrating that it is a novel inhibitor of NLRP3 that affects both classical and fatty acid NLRP3 triggers. AA counteracts the effects of both PA and nigericin on multiple intracellular signaling pathways, especially the JNK pathway. These mechanistic insights highlight pathways that may be targeted to delay or reverse the progression of sterile inflammation in many "lifestyle diseases". This work was supported by the Intramural Research Program of NIAID, NIH.

Session 4: Microbiology & Infectious Diseases

Yentli E. Soto Albrecht

University of Pennsylvania

Non-pathogenic variation in mitochondrial DNA modulates murine SARS-CoV-2 pathogenesis

Mitochondrial functional variants are ubiquitous in the population and represent differences in mitochondrial DNA (mtDNA). These variants are categorized into lineage-specific mitochondrial haplogroups and have been shown to contribute to differences in Human Immunodeficiency Virus CD4+ T cell levels and long-term prognosis in patients. However, the mitochondrial contribution to control of SARS-CoV-2 infection remains to be studied. Our lab previously generated mice containing murine mitochondrial haplogroups 129 or NZB mtDNA on the C57BL/6J (B6) nuclear background. The total mtDNA variance between groups is less than 100 base-pairs (bp) out of the 16.5 kbp mitochondrial genome. We chose to study the contribution of mitochondrial background to SARS-CoV-2 pathogenesis in the murine model expressing the human ACE2 viral receptor under the keratin 18 promoter (k18-hACE2). We generated mice on the k18-hACE2 nuclear background with B6 (wild-type), 129 or NZB mtDNA. Consistent with the literature, we found that k18-hACE2 mice with wild-type mtDNA exhibited 100% fatality when challenged intranasally with SARS-CoV-2. Remarkably, mice with 129 or NZB mtDNA both showed increased survival (40%) over wild-type. Importantly, mice with NZB mtDNA showed markedly decreased clinical disease at 6 days post infection compared to mice with 129 ($p = 0.0444$) and B6 ($p = 0.0027$) mtDNA. Cumulatively, these data demonstrate that mitochondrial haplotype alone can influence clinical disease in a murine model of severe SARS-CoV-2 infection. Studies on immune and viral correlates of protection are ongoing, as well as challenge of an alternative SARS-CoV-2 murine model to characterize the generalizability of this phenotype. Our discovery of the novel role mitochondrial haplotype plays in SARS-CoV-2 pathogenesis may contribute to increased understanding of host factors that impact the severity of COVID-19.

Umaru Barrie

University of Texas Southwestern

ERK1/2 Activation in Macrophages is Necessary for Efficient Leishmania amazonensis Internalization and Pathogenesis

Leishmania, an obligate intracellular protozoan parasite, binds several host cell receptors to trigger uptake by phagocytic cells, ultimately leading to visceral or cutaneous leishmaniasis. Upon engaging receptors on macrophages and other phagocytes, Leishmania activates a series of signaling pathways during its internalization process, which are critical for establishment and persistence of infection. Thus, preventing internalization could be a novel therapeutic strategy for leishmaniasis. However, the host machinery that mediates amastigote uptake is poorly understood. Here, using small-molecule inhibitors and primary macrophages lacking specific Mitogen-activated protein kinases/Extracellular signal regulated kinases (MAPK/ERK), we demonstrate that ERK1/2 mediates efficient Leishmania amazonensis uptake and beads phagocytosis by macrophages. Consistent with these results, MEK1/2- or ERK1/2-deficient macrophages are inefficient at amastigote uptake and produce large F-actin-rich phagocytic cups, showcasing a defective phagocytic process. In addition, Syk and Abl/Arg family kinases are necessary and mediate the ERK1/2 kinase activity that is required for efficient uptake. Finally, trametinib, a MEK1/2 inhibitor, significantly reduces disease severity and parasite burden in Leishmania-infected mice by reducing the number of internalized parasites and activating cytokines and chemokines to mount an inflammatory response to Leishmania. Our studies demonstrate that maximal Leishmania infection and efficient phagocytosis require MAPK/ERK family kinases, highlighting MAPK/ERK family kinase-mediated signaling pathways as potential therapeutic targets for leishmaniasis.

Soo hun Yoon

Michigan State University

Uncovering the role of ubiquitin-like modulation of 3’3’-cGAMP signaling in Vibrio cholerae El tor

Cyclic di-nucleotide second messenger molecules modulate global pathways imperative for cellular adaptation in bacteria and eukaryotes. cGAS in eukaryotes synthesizes 2’3’cyclic GMP-AMP (cGAMP) upon detection of DNA in the cytosol following viral infection or nuclear damage, to activate the STING inflammatory response and immune targeting of cancer cells. In Vibrio cholerae El tor, the main culprit of the current cholera pandemic, DncV synthesizes 3’3’-cGAMP following phage infection to activate CapV, a phospholipase that degrades the cell membrane, killing the infected cell as a mechanism of altruistic suicide to protect the remaining bacterial community. DncV and CapV are found within a cluster of genes dubbed cyclic di/oligonucleotide (cdN) based antiphage signaling system (CBASS). CBASS is found in all bacterial phyla. Understanding cGAS’s ancestral counterpart, DncV and other homologs, can lead to more effective vaccine adjuvants and exploitation of phage defense mechanisms for phage therapy. vc0180 and vc0181, encoding E1/E2-like ubiquitin activating and deubiquitinase domains, respectively, are encoded downstream of DncV and enhance the cdN response through an unknown mechanism. No ubiquitin-like modifier has been identified nor is the protein target/s of VC0180 and VC0181 known. I have identified an uncharacterized gene we name bumo (bacterial ubiquitin modifier) in the V. cholerae genome as the potential substrate of VC0180/181, as deletion of bumo phenocopies ∆vc0180/vc0181 leading to the absence of DncV. I discovered that VC0181 is a novel protease that degrades DncV as it is highly detected only in the absence of VC0181. Furthermore, I show that the presence of Bumo inhibits DncV degradation by VC0181. I am currently exploring the role of bumo and vc0180/181 in phage resistance, the regulation of CBASS components, and synthesis of 3’3’ cGAMP. My research is uncovering a novel ubiquitin-like mechanism of protein modification important for phage defense that is widespread in bacteria.

Hannah Bell

University of Rochester

The Function of Type Three Secretion System Protein VopZZ

Vibrio cholerae is an environmental Gram-negative bacterium with the potential to cause the diarrheal disease cholera. Strains are genetically diverse, while only O1 and O139 serogroup strains cause epidemic outbreaks, non-O1/non-O139 serogroup strains are a significant cause of sporadic disease worldwide and employ various virulence factors. A recently identified pathogenicity island encoding a type three secretion system (T3SS), present in ~40% of non-O1/non-O139 serogroup strains, is required for colonization and disease in animal models of infection. The T3SS acts as a molecular syringe to inject proteins, called effectors, directly into a host eukaryotic cell. VopZZ, one of the 13 known effector proteins encoded in T3SS+ V. cholerae, is essential for in vivo colonization and bacterial adherence, and host cytotoxicity in vitro. Despite the importance of VopZZ in V. cholerae infection, the molecular function of VopZZ remains unknown. We present evidence that a C-terminal 23-residue sequence is a functional domain required for in vitro mammalian cell adherence and cytotoxicity. The sequence encodes a predicted amphipathic alpha-helix with similarity to the styelin family of antimicrobial peptides. Regions of VopZZ examined include those corresponding to [Vp] VopZ-mediated inhibition of a host MAP kinase (TAK1) important for innate immune signaling, and an RGD motif unique to V. cholerae VopZZ that may serve as an integrin ligand if exposed on the host cell surface. The putative TAK1 domain and RGD motif were dispensable for V. cholerae mediated cytotoxicity, indicating that these domains may encode other functions to facilitate colonization or disease. These collective results suggest that the VopZZ styelin motif plays a critical role in T3SS-mediated V. cholerae adherence to and derangement of the host cell membrane.

Poster Session I

Friday July 8th, 7:00-8:00 PM, Kokopelli's Trail

1) Jerome Arceneaux

Vanderbilt University

Mapping the Cellular Composition of Resected Cortical Tubers and Perituberal Tissues

2) Ryan Reyes

University of Texas Health San Antonio

CD122-targeted interleukin-2 and αPD-L1 Treat Bladder Cancer and Melanoma via Distinct Mechanisms Including Tissue-selective γδ T cell Activation and CD122-driven Natural Killer Cell Maturation

3) Khoa Nguyen

Tulane University

LKB1-SIK1 Signaling Regulates the Cancer Stem Cell Axis in TNBC

4) Lauren Morehead

University of Arkansas for Medical Sciences

Harnessing resveratrol-mediated Restoration of MHC-I in Melanoma to Enhance Immunotherapy

5) Luke Brennan

Indiana University

Perspectives on Cervical Cancer Screening Technologies from Indiana Healthcare Providers

6) Alexandra A. Miller

University of Texas Health Science Center, Houston

Systematic Review of Aptamer Sequence Reporting in the Literature Reveals Widespread Unexplained Sequence Alterations

7) Chloe Gao

University of British Columbia

Understanding the mental health and recovery needs of first- and second-generation Canadian East Asian immigrant youth aged 12–24: A Strategy for Patient-Oriented Research (SPOR) collaboration study

8) Andres Dajles

University of Iowa

The Zero Value: A New Approach to Quantifying the Importance of an Effect

9) Alexander Girgis

Johns Hopkins University

Distinct Adaptive Immune Receptor Repertoires Characterize Post-Treatment Lyme Disease

10) Saif Yasin

University of Maryland

Characterizing the Mechanism of HIV-1 Genome Dimerization

11) Andy An

University of British Columbia

Unresolved Immune Dysfunction is Lethal in Both COVID-19 and non-COVID-19 Sepsis

12) Frances Li

University of Colorado

Defining MARCO-virus Interactions Important for Alphavirus Clearance from the Circulation

13) Savannah Taylor

University of Texas Southwestern

Colonocyte-derived lactate promotes E. coli fitness in the context of inflammation-associated gut microbiota dysbiosis

14) Meryem OK

University of North Carolina at Chapel Hill / North Carolina State University

A novel platform using healthy human colonic epithelium demonstrates the impact of Clostridioides difficile toxins on colonic epithelial integrity

15) Chloe Cavanaugh

Rutgers Robert Wood Johnson Medical School / Princeton University

Mechanism and Therapeutic Implications of Host Cell Telomerase Modulation by Human Cytomegalovirus

16) Kyler Crawford

Medical College of Wisconsin

Structural insights on the 'DARC' side of CXCL12 signaling and bone marrow metastasis

17) Andrew Oleksijew

University of Nebraska

Fluid stasis promotes dysregulated mechanosensory signaling pathways in cholestatic disease

18) Michaela Cooley

Case Western Reserve University

Nanobubble Contrast-Enhanced Ultrasound Imaging for Assessing Tumor Vascular Permeability and Nanoparticle Extravasation

19) Gary Ge

University of Rochester

Power laws in medical imaging

20) Dane Sessions

University of Virginia

Opa1 opposes mitochondrial fission to maintain ETC assembly and function in KRas-mutant lung adenocarcinoma

21) Melissa Anfinson

Medical College of Wisconsin

The novel MYH6-E1584K tail domain variant associated with hypoplastic left heart syndrome impairs cardiomyocyte mechanics in vitro

22) Aditi Misra

University of Rochester

Transcriptomic Analysis of Neonatal Cardiac Regeneration and Scar Resolution

23) Alexandra Vik

University of Maryland

Role of macrophage polarization in TLR4-SNP-associated increased colitis severity

24) Brandon Hubbard

Yale School of Medicine

A novel mass-isotopomer method assesses hepatic electron transport chain and non-canonical anaplerotic fluxes in vivo

25) Anne Skelton

Virginia Commonwealth University

Are Osteoblast-derived Matrix Vesicles a Species of Exosome?

26) Johnathan Gigas

University of Rochester

Longevity-associated regulation of SIRT6 by AMPK alters DNA binding and interacting partners

27) Roger Zou

Johns Hopkins University

Characterization of genome editing dynamics with multi-target CRISPR

28) Valentyna Kostiuk

Yale School of Medicine

The role of nucleoporin 107 in early embryonic patterning

29) Matthew Miller

University of Iowa

Cdkn1a (p21) promotes skeletal muscle atrophy by inhibiting cyclin-dependent kinase 1 (Cdk1)

30) Alex Moauro

Michigan State University

Exogenous Oct4/Pou5f1 is not required to produce induced extraembryonic endoderm stem cells during somatic cell reprogramming

31) William Tang

University of Utah

MicroRNA-155 Expression in T cells Orchestrates Immune Responses in Colon Cancer

32) Jayden Bowen

University of Iowa

The role of TREM-1 in allergic airway inflammation

33) Emily King

University of Colorado

Acute lung inflammation is characterized by early recruitment of monocytes to the airspace and continued recruitment to the pulmonary interstitium

34) Jonathan Liang

Yale School of Medicine

Saturated fatty acid activation of the NLRP3 inflammasome depends more strongly on JNK activation than on ROS

35) Andrew Griswold

Weill Cornell/Rockefeller/Sloan Kettering Tri-Institutional MD-PhD Program

The NLRP1 Inflammasome Induces Pyroptosis in Human Corneal Epithelial Cells

36) Rose Caston

University of Utah

Suppression of broad-band gamma associated with thermo-nociception in the posterior insula

37) Corey Shayman

University of Utah

The Effects of Age on Sensory Integration during Navigation

38) Alexander Baez

Stony Brook University

Striatal Cholinergic Interneurons are Driven to Increased Acetylcholine Output in the SAPAP3-deletion Model of Obsessive Compulsive Disorder

39) Rachel Furhang

State University of New York Downstate Health Sciences University

A Single Traumatic Bain Injury Leads to Chronic, Progressive White Matter Atrophy and Increased Phosphorylated Tau Expressing Oligodendrocytes

40) Anthony Chesebro

Stony Brook University

Characterizing neurogliovascular unit response to natural sensory input

41) Tyler Waltz

Medical College of Wisconsin

Peripheral Neuron Excitability is Mediated by Schwann cells in Fabry disease

42) Edith Hernandez

University of Minnesota

Repurposing Metformin as a treatment for Cocaine Use Disorder via AMPK Activation

43) Thao Le

Vanderbilt University

Liver Fibroblast Growth Factor 21 (FGF21) is Required for the Full Body Weight- and Appetite-Suppressing Effects of Glucagon-Like Peptide-1 (GLP-1) Receptor Agonism

44) Mark Mizrachi

Donald and Barbara Zucker School of Medicine at Hofstra/Northwell

HMGB1-mediated microglial activation as a mechanism for cognitive dysfunction in neuropsychiatric lupus

45) Frances Zakusilo

University of Rochester

Comparative Study of Oxidative Stress Resistance in Naked Mole Rat, Mouse, Degu, and Human Astroglia

46) Bilal Moiz

University of Maryland

Niemann-Pick Disease Type C1 inhibition leads to loss of barrier integrity and altered metabolic phenotype in brain microvascular endothelial cells

47) Joseph Vecchi

University of Iowa

Feature Characteristics and Signaling Mechanisms Involved in Spiral Ganglion Neurite Guidance

48) David Freeman

University of Utah

CRIPTO regulates fibroblast-tumor crosstalk and cell-state in breast cancer

49) Xue Qi “Amy” Wang

University of British Columbia

Targeting SS18-SSX biology in synovial sarcomagenesis

50) Matthew Loberg

Vanderbilt University

Deciphering the role of cancer-associated fibroblasts in thyroid cancer progression

51) Tejaswini Reddy

Texas A&M School of Medicine

NOS inhibition augments PI3K inhibitor efficacy in metaplastic breast cancer.

52) Neville Dusaj

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Single-cell multi-omics of human clonal hematopoiesis reveals that DNMT3A R882 mutations perturb early progenitor states through selective hypomethylation

53) Chad VanSant-Webb

University of Utah

Dissecting the role of Inflammation and miR-146a in Hepatocellular Carcinoma

54) Justin Magrath

Tulane University

Transcriptome Analysis Identifies SRC Kinase Inhibitors as a Therapeutic for Desmoplastic Small Round Cell Tumor Cancer Stem Cells

55) Nisitha Sengottuvel

University of North Carolina at Chapel Hill

Lineage Switching and Tumor Microenvironment Roles of Tumor Suppressors in Autochthonous Lung Cancer Models

56) Connor Hughes

University of Colorado

Eya3 regulates NF-kB signaling and tumor progression in Triple Negative Breast Cancer

57) Ayana Jamal

Medical College of Wisconsin

Investigating the role of shroom3 in cardiac function and regeneration

58) Nickole Moon

University of Maryland

Extracellular vesicles coordinate stress-dependent mitochondrial programming

59) Redwan Bhuiyan

University of Connecticut

Dissecting the genetic programming of inflammatory cytokine responses in human islets

60) Eudorah Vital

Emory University School of Medicine / Georgia Institute of Technology

Linking Circulatory Turbulence and the Pathophysiology of Sickle Cell Disease (SCD)

Poster Session II

Saturday July 9th, 4:00-5:00 PM, Kokopelli's Trail

1) Juan Santiago

University of Colorado

Color Representation in Primary Visual Cortex

2) Susie Turkson

Virginia Commonwealth University

Fear Extinction Deficits in Women Living with HIV and PTSD

3) Justin Johnson

Yale School of Medicine

COVID Coronary Vascular Thrombosis: Correlation with Neutrophil but not Endothelial Activation

4) Gregory Chen

University of Pennsylvania

Tara the T-cell's Big Adventure: Scientific Communication through a Children's Book on Chimeric Antigen Receptor T-cell Therapy

5) Sijin Zheng

Yale School of Medicine

Development of novel fetal hemoglobin inducers using targeted protein degradation

6) Emily Lubin

University of Pennsylvania

De novo Mutations in Replication-Independent Histone Genes Elude Diagnosis by Exome/Genome Sequencing

7) Carol Deaton

University of Rochester

Presenilin 1 Modulates Vacuolar Function and Tau Degradation

8) Nadia DiNunno

Pennsylvania State College of Medicine

The structure of mammalian derived HBV capsid in complex with a fluorescent HAP13 derivative

9) Austin Jolly

University of Colorado

The chromatin remodeler Brg1 regulates adventitial progenitor-to-myofibroblast differentiation and *in situ* vascular fibrosis

10) Daniel Nguyen

University of Louisville School of Medicine

The role of TAK1 in cardiac fibroblast activity

11) Alecia Alto

The University of Alabama at Birmingham

Expression of Human Endogenous Retrovirus Envelopes in Latent HIV Infection

12) Thomas Forman

University of Colorado

Srsf3-mediated alternative RNA splicing downstream of PDGFRa signaling in craniofacial development

13) Oliver Culver

Medical University of South Carolina

Adaptive and predictive valence encoding patterns of opioid sensitive cholecystokinin dopamine neurons during a conditional classical pavlovian task

14) Rohit Singla

University of British Columbia

The Kidneys Are Not All Normal: Speckle Distributions of Transplant Kidneys

15) Zachary Billman

University of North Carolina at Chapel Hill

Identification of translocated effectors in Histoplasma capsulatum

16) Joan Shang

Icahn School of Medicine at Mount Sinai

Dissecting the Gut Microbiota for Immune Checkpoint Blockade (ICB) – Resisting Microbes and Exploring the Generalizability of Microbiota-ICB Studies

17) Lianne Cho

University of British Columbia

Psychometric properties of the Beck Depression Inventory using classical test theory and Rasch analysis

18) Nathaniel Skillin

University of Colorado

Collective cell mechanosensing drives muscle myotube alignment on monodomain liquid crystal polymer networks

19) Alec Xiang

Donald and Barbara Zucker School of Medicine at Hofstra/Northwell

Extracellular Vesicles as Markers of Blood-Brain Barrier Dysfunction in Systemic Lupus Erythematosus

20) Ryan Kawalerski

Johns Hopkins University

tRNA decay is rapid and coupled with mRNA decay

21) Dana Oakes

University of Louisville School of Medicine

Establishing an animal model of cerebral/cortical visual impairment (CVI)

23) Loren Oh

University of North Carolina at Chapel Hill

Provider Communication and HPV vaccine uptake: A meta-analysis and systematic review

24) Hannah Kondolf

Case Western Reserve University School of Medicine

An inducible system for the study of differential gasdermin family member functions

25) Cuilee Sha

Stony Brook University

A Titratable Mouse Model of Necrotizing Enterocolitis

26) Kelsey Kines

University of Colorado

The effect of Semaphorin7A on postpartum and triple-negative breast cancer metastases and chemoresistance.

27) Sam LaMagna

State University of New York Upstate Medical University

The Role of Interphotoreceptor Coupling in Mouse Visual Sensitivity Measured with an Operant Behavioral Assay

28) Christine Rafie

University of Miami

Reinvigoration of Exhausted T Cells with Non-coding RNAs in Pancreatic Ductal Adenocarcinoma

29) Michelle Culbertson

University of Utah

Evolutionary conflicts between poxviruses and bat immunity

30) Ryan Rebernick

University of Michigan

Carcinoma of Prostate Sequencing of Tumor and cliNicalEndpoints (CAPSTONE): Leveraging genomics to understand clinical outcomes in advanced prostate cancer

31) Katherine Gillis

University of Utah

FoxA1/2 regulate the epigenetic landscape of NKX2-1-negative lung adenocarcinoma to facilitate cancer cell lineage switching

32) Matthew Hadiono

Medical College of Wisconsin

Modulation of gut microbial metabolism and energy expenditure by xenobiotics and bacterial metabolites

33) Briaunna Minor

University of Rochester

Estrogen Modulation of Infiltrating Polymorphonuclear Cells Promote Tumor Progression in a Model for Lymphangioleiomyomatosis (LAM)

34) Omar Moustafa

University of Connecticut

Novel Single Nuclear Analysis Approach to Study BBB Endothelial RNA Regulation in Dementia

35) Federico Prokopczuk

University of Alabama

Impact of COVID-19 Treatment Regimens on Fungal Growth and COVID-Associated Secondary Mold Infection

36) Wesley Huang

University of Michigan

Defining the role of ferroptosis in inflammatory bowel disease (IBD)

37) Hei-Yong Lo

University of Colorado

Investigating the importance of ASPM RNA at the centrosome on cell cycle division

38) Franklin Ning

University of Maryland

Elucidating immune cell interactions to enhance autologous monocytes and dual-interferons as a therapy for ovarian cancer

39) Mitchell Lefebvre

University of Iowa

Kupffer cells mediate rapid recruitment of protective circulating memory CD8 T cells during microbial infection of the liver

40) Cassandra Woolley

University of Louisville School of Medicine

Regulation of cell-surface expression of the Fc Receptor for IgM (FcMR) in human lymphocytes

41) Laurel Darragh

University of Colorado

Elective nodal irradiation mitigates local and systemic immunity generated by combination radiation and immunotherapy

42) Skylar Wright

University of Connecticut

Elucidating the role of metabolites in the immune response to pyroptosis

43) Shuyang Qin

University of Rochester

“Reverse abscopal effect”: Systemic immunosuppression and immunotherapy resistance in metastatic melanoma

44) Safwan Elkhatib

University of Nebraska

T-Lymphocyte Tyrosine Hydroxylase Regulates TH17 T-lymphocyte Profiles in Psychosocial Trauma Independent of Mitochondrial Redox

45) Adam Wegman

SUNY Upstate Medical University

Monomeric IgA1 Antagonizes IgG1-Mediated Antibody-Dependent Enhancement of DENV-3 Infection

46) Ronan Talty

Yale School of Medicine

Preclinical and clinical ferroptosis inducers potentiate checkpoint inhibitor immunotherapy

47) Brenda Seymour

University of Colorado

The Bacterial Metabolite Indole Promotes Collagen Induced Arthritis through Enhanced Th17 Immunity

48) Likhitha Kolla

University of Pennsylvania

Time of clinic appointment and advance care planning discussions in oncology

49) Emilie Fisher

Vanderbilt University

Improving anti-tumor CD8+ T cell function through manipulating glutamine metabolism

50) Alexa Blanchard

University of Maryland

The role of mast cells in shaping neonatal hippocampal development

2022 DIVERSITY TRAVEL AWARDEES

The following students received a Diversity Travel Award to this conference:

Oluwamayokun Oshinowo
Emory University

Bojing Jia
University of California San Diego

Javier Eli Sierra Pagan
University of Minnesota

Mary Adeyeye
University of Texas Houston

Umaru Barrie
University of Texas Southwestern

Ryan Reyes
University of Texas Health San Antonia

Khoa Nguyen
Tulane University

Tyrone DeSpenza
Yale School of Medicine

Jerome Arceneaux
Meharry Medical College/Vanderbilt University

Lauren Morehead
University of Arkansas

SUNDAY MORNING ACTIVITIES

Copper 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. Transportation will be provided.

Officers Gulch: Our short hike is a relaxed stroll known for beautiful lake views and opportunities to spot various Colorado wildlife. This hike has a modest elevation gain of 100 ft and a round trip distance of 1.7 miles.

Mayflower Gulch: Our long hike is known for having abundant wildflowers. This hike has a more challenging altitude gain is 1450 ft and a round trip distance is 3.5 miles.

Alpine Fishing*

Spend the day fishing the beautiful and accessible black lakes area for trout with our two student guides. Rod and reel (fly and spinning/conventional) provided, although spots are limited.

Participants must buy a Colorado fishing license a non-resident or resident license for 1 day online at https://www.cpwshop.com/licensing.page. Colorado Residents pay $14.46 and Non-residents pay $17.65. Transportation to and from the fishing area will be provided. Participants will be asked to show their licenses before departing.

Copper Activities Pass*

For $79, participants can purchase an Activity Day Pass for Copper's many summer activities including: unlimited scenic chair rides, unlimited boating activity sessions and attempts on the Climbing Wall, 2 runs through the Woodward WreckTangle, 1 ride on the Rocky Mountain Coaster, 2 sessions on the Bungee Trampoline, 1 ride on the Go-Kart track and 2 rides on the Zip Line.** Single activity passes can also be purchased for $10-$35 at ticket windows for each activity.

To purchase a pass and for more details visit https://www.coppercolorado.com/things-to-do/activities-amenities/summer-activity-passes .

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!

**Fly fishing or participation in Copper activities pass will require an additional cost.*

*** 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.*

We thank our major sponsors:

CU Department of Medical Oncology
CU Cancer Center
CU Department of Medicine
CU Department of Radiation Oncology

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