SCHOOL OF MEDICINE

Medical Scientist Training Program

UNIVERSITY OF COLORADO **ANSCHUTZ MEDICAL CAMPUS**

Second Annual Retreat

February 28th, 2020

Nighthorse Campbell Building

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Schedule

TIME	SESSION
8:00-8:45AM	Registration *Coffee and light breakfast available* *Students please put up posters at this time*
8:45-9:00AM	Opening Remarks
9:00-9:30AM	State of the Program Address
9:30-10:30AM	Student Talks: *Meghan Kellett, Leah Bowen, Andy Tekriwal, Kelly HIga*
10:30-10:45AM	1 minute student talks
10:45-11:45AM	Poster Session 1
11:45-12:30PM	Lunch
12:30-1:30PM	Keynote: Dr. Simon Hambidge, MD/PhD Class of 1994
1:30-2:30PM	Student Talks: *Jason Silver, Anagha Inguva, Humphrey Petersen-Jones, Cecilia Levandowski*
2:30-4:00PM	Break-out sessions (*follow instructions on badge for room rotation)*
	*Case Study in Happiness: Lessons from Child Neurology Residency *Dr. Timothy Bernard, Adam Finney and Dr. Dylan Brock*
	*Building a personal brand using social media *Storm Gloor, MBA*
	*Discovering Career Opportunities as an MD/PhD: A conversation with CU MSTP alumni *Dr. Brian Harry ('15), Dr. Benjamin Young ('92)*
4:00-5:00PM	Poster Session 2
5:15-5:30PM	Awards and Retreat Conclusion
	Post-Retreat Celebration. Location: Stanley Beer Hall

Visit our website (www.mstp-retreat.co) for directions to the Stanley Beer Hall for our post-retreat celebration.

Keynote

We are thrilled to have Dr. Simon Hambidge, CU MSTP class of 1994, as our keynote speaker for the 2020 MSTP retreat. Dr. Hambidge received his PhD in Microbiology and Immunology in the laboratory of Dr. Peter Sarnow and then went on to complete a residency in Pediatrics with a Primary Care Faculty Development Fellowship and a certificate in Public Health at the University of Colorado. Dr. Hambidge then served as the Director of General Pediatrics for Community Health Services at Denver Health for 8 years before becoming the Chief Executive Officer of Denver Community Health Services (DCHS) and Chief Ambulatory Officer at Denver Health in 2014. As DCHS CEO, Dr. Hambidge oversees a network of 9 community health centers and 18 school-based clinics, which provide health care to over 176,000 patients, a quarter of the Denver population. Dr. Hambidge is a leader in the Denver healthcare community and serves on the Board of Directors of the Denver Health Medical Plan, Colorado Community Health Network, Community Health Association of Mountain and Plains States, Reach Out and Read Colorado, and Medical Services Board of the state Medicaid agency.

Break-out Sessions

Case Study in Happiness: Lessons from Child Neurology Residency

Timothy Bernard, MD, MSCS: Child Neurology Residency Program Director

I am the father of 15-year-old twins, Jack and Sawyer. My kids were born at University Hospital on the same floor where I was born, which makes me a native of Colorado (a rarity these days). I specialize in pediatric stroke, which I like because most of my patients recover very well from their event, and it is a relatively new field. I enjoy road biking, golfing and skiing, when not driving my twins around Denver. I greatly enjoy working with medical students and young physicians to facilitate their personal and professional growth, which is the reason being program director is the best part of my job!

Adam Finney, MS: Child Neurology Residency Program Education Coordinator

I was born and raised in Colorado. I spent a few years in Texas but returned to Colorado as soon as I could. I have been in my role for the past 5 years and enjoy the people and the culture of the program. I spend my free time with my wife, two kids, and black labrador. We enjoy exploring all that Colorado offers including the mountains, brew pubs, wineries, music scene, and the Arts.

Dylan Brock, MD: Child Neurology Resident PGY5

I received my Undergraduate degree in Neural Science and Philosophy of Mind from New York University. I completed my master's degree and medical school at the University of Louisville.

Building a Personal Brand Using Social Media

Storm Gloor, MBA: Music Business Professor - CU Denver

Storm Gloor is an associate professor in the Music and Entertainment Industry Studies department of the College of Arts and Media at the University of Colorado Denver. He is the recipient of the university's 2018 Excellence In Teaching award. In 2014, professor Gloor developed and instructed what is considered to be the first Music Cities higher education course. Along with that course, professor Gloor teaches Music Marketing and oversees the internships for the College of Arts & Media. As part of the First Year Experience program at CU Denver, he teaches a course on the Beatles. Professor Gloor is also a Faculty Fellow in the Center for Faculty Development, is the immediate past president of the Music and Entertainment Industry Educators Association, and serves as a city councilman for Glendale, Colorado. He has presented at numerous events and programs, including SXSW.edu, South By Southwest Music, the Music Cities Convention, the MEIEA Music Educators Summit, the Future of Music Summit, the Underground Music Showcase, the Denver Music Summit, and the EdMedia world conference.

Discovering Career Opportunities as an MD/PhD: A Conversation with CU MSTP Alumni

Brian Harry, MD/PhD: Class of 2015, Assistant Professor Department of Pathology

Brian is Assistant Professor of Pathology at the University of Colorado School of Medicine and Medical Director of Special Chemistry and Referral Testing for University of Colorado Hospital Clinical Laboratories. In his current role he is developing clinical assays using liquid chromatography-mass spectrometry (LC-MS) methods to measure small molecules. His research aims to understand proteome dynamics and protein post-translational modifications in cancer, with a long-term vision to bring proteomics capabilities to the clinical lab.

Brian is a University of Colorado MSTP alumnus who completed graduate studies in cell death with Ding Xue, Ph.D. in the Department of Molecular, Cellular, and Developmental Biology at CU Boulder. In 2015 Brian joined the Clinical Pathology Residency Program at Massachusetts General Hospital in Boston, MA. During this time, he conducted postdoctoral work with Steve Gygi, Ph.D. at Harvard Medical School using proteomics to understand mechanisms of protein stability and degradation. He was also a Partners Healthcare Innovation Fellow and Consulting Senior Associate at Third Rock Ventures where he led efforts to ideate and build biotech companies, including Cedilla Therapeutics. Brian continues his work in biotech innovation and is currently Chief Laboratory Officer of SummitDx, a seed-stage company developing saliva liquid biopsy tests for head and neck cancer.

Benjamin Young, MD/PhD: Class of 1992,
Senior Global Medical Director for ViiV Healthcare

Dr. Benjamin Young, MSTP Class of 1992, received his PhD in the Chemistry and Biochemistry department at Boulder with Dr. Thomas Cech and then completed residency in Internal Medicine and a fellowship in Infectious Disease both at the University of Colorado. Since then he has held a number of leadership positions and has continued to pursue clinical research in HIV. Among his numerous honors he has been awarded "Denver's Top Doctors" by 5280 magazine as well as "Outstanding HIV/AIDS Clinician and HIV Leadership Award" from TheBody.com a group dedicated to improving care and quality of life for those with HIV. Dr. Young educates physicians on HIV medicine worldwide, with experiences in Russia, Ukraine, Uzbekistan and The Netherlands. Currently Dr. Young is the Senior Global Medical Director for ViiV Healthcare, a group dedicated to innovative approaches to the challenges of HIV.

One-Minute Talks

1. Brigit High, class of 2016
2. Elijah Christensen, class of 2014
3. Connor Hughes, class of 2016
4. Grant Lo, class of 2017
5. Amelia Burch, class of 2017
6. Laurel Darragh, class of 2017
7. Austin Jolly, class of 2016
8. Sarah Haeger, class of 2012
9. Michael Nash, class of 2016
10. Ricardo Villarreal, class of 2013
11. Taylor Soderborg, class of 2012

Student Presentations

Meghan Kellett, class of 2016, Schweppe lab

Late stage thyroid cancers characterized by metastasis and invasion have a poor prognosis compared to those with localized disease. However, there are limited therapeutic options and few biomarkers to indicate which patients will develop aggressive disease. Our lab has identified Focal Adhesion Kinase (FAK) as a key regulator of thyroid cancer growth, invasion, and metastasis. FAK is a non-receptor tyrosine kinase that is auto-phosphorylated at tyrosine 397 (Y397) in response to integrin or growth factor receptor signaling resulting in the activation of downstream signaling pathways. FAK has also been shown to localize to the nucleus in response to cellular stress via a nuclear localization sequence to promote increased cell survival. We have found that FAK is localized to the nucleus in a subset of thyroid cancer patients, but it's unclear how FAK is localizing to the nucleus and what its function is in the nucleus. Given the role of cellular stressors to promote tumor growth and metastases in thyroid cancer, I hypothesize that cellular stress induces nuclear localization of FAK to promote a more aggressive phenotype in thyroid cancer. I first analyzed the role of hypoxia since low oxygen environments cause tumor cells to secrete pro-inflammatory cytokines to recruit blood vessels to stimulate tumor growth. I found that FAK localizes to the nucleus within 30 minutes of exposure to a hypoxic environment of 1% oxygen. Furthermore, I found that this nuclear localization is dependent on phosphorylation of Y397 FAK. Next, I addressed the functional role of FAK in the nucleus and found that thyroid cancer cells secrete high levels of pro-angiogenic cytokines that is reduced when FAK is excluded from the nucleus in hypoxia. Thus, hypoxia induces nuclear localization of FAK through phosphorylation of Y397 FAK to promote the secretion of pro-angiogenic cytokines. Overall, nuclear FAK may serve as a biomarker of aggressive disease and novel therapeutic target in thyroid cancer.

Leah Bowen, class of 2015, Rentschler lab

Microtexture contact mechanics is a growing area of research that arose from study of the remarkable adhesive properties of geckos. The microscopic features on gecko toe pads and bio-inspired fabricated microtextures have varying adhesion and friction properties relative to smooth surfaces. Here, we investigate the friction properties of soft silicone microtextures with soft tissue or tissue analog substrates.

Coefficient of friction was determined using a custom traction measurement platform. Strained soft microtextures, stiff microtextures, and smooth silicone were studied against a soft polyvinyl chloride tissue analog with and without a mineral oil lubricant. For unlubricated samples, smooth silicone friction does not trend with strain while both soft and stiff texture friction increases with strain. Unlubricated smooth silicone has the largest coefficient of friction and stiff textures the smallest. With mineral oil lubrication, smooth silicone still does not trend with strain, but soft and stiff texture friction now display a decrease in friction coefficient with increasing strain. Lubricated smooth silicone has the smallest coefficient of friction and stiff textures have the highest.

A finite element model was developed to elucidate the mechanisms behind these phenomena. A two-dimensional planar model was constructed using a cohesive zone model for unlubricated friction and no cohesive zone model for lubricated friction. A PVC sled with rigid backing was indented and sheared against strained soft or stiff microtextures.

Microtextures were added to endoscopy balloons and their peak anchoring force was measured in ex-vivo porcine small intestine. Textures included cylindrical patterns of different materials, sizes, locations, and aspect ratios, ribbed, and conical. Stiffer materials, low aspect-ratio textures, and increased texture coverage contribute to larger peak force. Smooth latex balloons, used for standard enteroscopy, have the lowest overall peak force.

Andy Tekriwal, class of 2015, Felsen and Thompson labs

Parkinson's disease (PD) is a progressive neurodegenerative disorder caused by loss of dopaminergic neurons (DANs) in the basal ganglia (BG). The link between loss of DANs and onset of PD motor symptoms is well substantiated; however, the degree of impairment depends on the context of the movement (e.g., level of motivation, degree of certainty). Although well documented in the clinical literature, the underlying neural mechanism leading to some movements being more impaired than others is not well understood. To investigate, we record neural activity from BG output nuclei of hemi-PD and control mice trained on a two-alternative forced choice task. We elicited unilateral DAN loss via 6-hydroxydopamine infusion into the left substantia nigra pars compacta. To acquire neural recordings, we implanted drivable tetrodes into a principal output nucleus of the BG, the substantia nigra pars reticulata (SNr), ipsilateral to DAN loss. Control mice were similarly implanted. We compared behavior and neural activity between two conditions requiring otherwise-equal orienting movements: stimulus-guided or internally-specified. Under the stimulus-guided condition, the direction of movement (left vs. right) was selected based on the identity of the stimulus. Under the internally-specified condition, the direction of movement was selected based on recent history of rewarded movements (left vs. right). Blocks of stimulus-guided and internally-specified trials were interleaved within the behavioral session, allowing for within-session comparisons of behavior and neural activity between the two conditions. We hypothesized that the BG differently process stimulus-guided and internally-specified movements. Consistent with this hypothesis, we found that DAN loss led to greater behavioral impairment on internally-specified trials than on stimulus-guided trials.

Kelly Higa, class of 2012, DeGregori lab

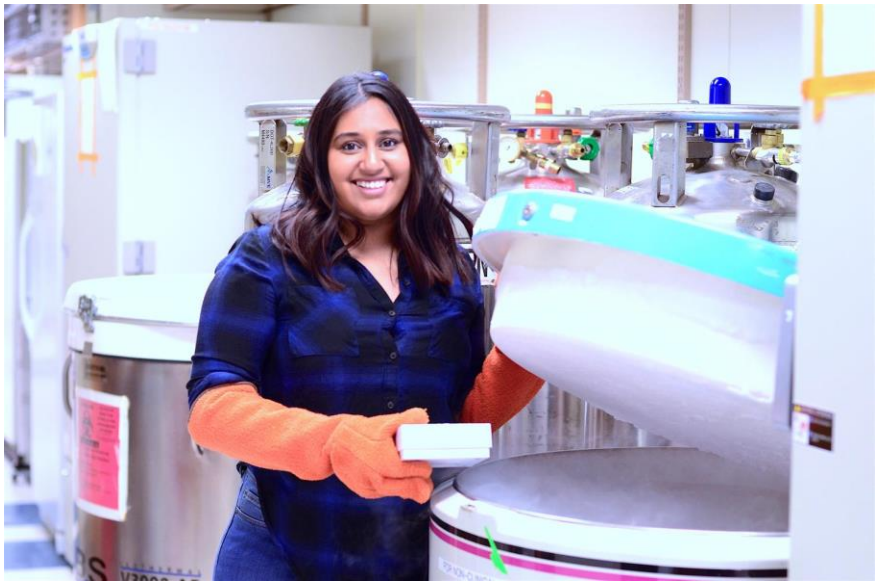
Clonal hematopoiesis (CH), myelodysplastic syndrome (MDS), and acute myeloid leukemia (AML) are associated with aging and prior chemo/radiotherapy, which in turn are associated with chronic inflammation. The Adaptive Oncogenesis Model proposes that a context that perturbs homeostasis can select for phenotypes that are adaptive in the perturbed context. Chronic exposure to the pro-inflammatory cytokine interleukin-1 (IL-1) impairs normal hematopoiesis by inducing precocious myeloid differentiation at the expense of hematopoietic stem and progenitor cell (HSPC) maintenance. Cebpa is a transcription factor essential for myeloid differentiation and is frequently mutated or downregulated in AML. We hypothesized that because chronic IL-1 causes precocious myeloid differentiation, it drives selection for mutant HSPC clones that block myeloid differentiation, such as through Cebpa loss of function. To test this, we competitively transplanted HSPC from Cebpa wild-type (WT) or HSPC-specific knockout (KO) donors into recipients mildly conditioned with busulfan, and treated mice with or without IL-1 for 20 days. While in the absence of IL-1 WT and KO HSPC maintained similar levels of chimerism in all hematopoietic compartments, IL-1 selectively drove expansion of KO multipotent progenitors (MPP), particularly the myeloid-biased MPP_3. RNA-Seq following competitive transplants revealed that while IL-1 drove upregulation of genes related to cell cycle, cellular metabolism, and protein synthesis in WT MPP_3, KO prevented these changes. Further, KO prevented IL-1-driven upregulation of myeloid differentiation genes and downregulation of HSC genes. Cell cycle analysis functionally validated that KO protected MPP_3 from IL-1-driven cell cycle entry. Finally, while IL-1-exposed WT MPP_3 had reduced colony-forming ability, KO MPP_3 were unaffected by IL-1 exposure.

Our data support a model in which Cebpa KO prevents chronic IL-1 from inducing differentiation of MPP_3, resulting in a more quiescent, stem-like phenotype. Our data provide an example of somatic evolution in the bone marrow where chronic IL-1 drives selection for HSPC clones harboring mutations that counteract the negative effects of chronic IL-1. These findings highlight the importance of chronic inflammation in the early events that drive development of CH, MDS, and AML.

Jason Silver, class of 2013, Anseth lab

Skeletal muscle has remarkable regenerative capacity, but muscle microenvironment often stiffens over time as a result of aging, injury, or disease. Each muscle fiber is surrounded and directly linked to an extracellular matrix (ECM) that possesses inherent elasticity due to the presence of crosslinked proteins. While initially thought to be a passive structure, the ECM actively participates in biochemical and physical signaling to facilitate the resident skeletal muscle stem cells (MuSC) orientation, expansion, and differentiation, all affecting the regenerative capacity of muscle. The impact of muscle stiffness on the regenerative behavior of MuSCs remains elusive. Here, we show that muscle elasticity increases after injury and persists beyond myofiber regeneration, maintaining highly proliferative and activated MuSCs. To model the transforming muscle niche, a dynamic hydrogel system was developed to stiffen in the presence of myoblasts. Gels were formed using a SPAAC chemistry where poly (ethylene glycol) monomers functionalized with dibenzocyclooctyne (DBCO), a ring-strained alkyne, can spontaneously react with azide moieties. Polymer networks with excess DBCO moieties can undergo a secondary radical-mediated crosslinking upon light exposure with a photoinitiator, stiffening the polymer. Using these hydrogels, we found proliferation and migration of both myoblasts on hydrogels and MuSCs on explanted myofibers embedded into matrices were dependent on stiffness, in agreement with our in vivo observations. Nuclear-cytoplasmic shuttling of both YAP and TAZ, which comprise part of the cell's mechanotransduction machinery, transduces the differences in matrix elasticity into the MuSC proliferative responses. Thus, these mechanosensitive transducers may contribute excessive MuSC proliferation within the post-injury or diseased muscle.

Anagha Inguva, class of 2016, Jordan lab



AML is an aggressive disease with low cure rates, due to ineffective eradication of leukemia stem cells (LSCs) with standard therapy. The recently FDA approved venetoclax, a BCL2 inhibitor, with azacitidine, a hypomethylating agent leads to a 70% response rate in AML patients compared to 10% with conventional therapy. Analysis of patients treated with this regimen showed direct targeting of LSCs. Previous work has shown that LSCs have a unique dependence on oxidative phosphorylation (OXPHOS) for energy production, and this can be perturbed by BCL2 inhibition. However, the connection between BCL2 and OXPHOS remains unclear. One unexplored function of BCL2 in LSCs is its role in calcium regulation. BCL2 regulates calcium flux between the ER and mitochondria by activating or deactivating calcium channels at the ER and mitochondria. BCL2s regulation of calcium flux between the mitochondria and ER is crucial for LSC survival, as LSCs rely on OXPHOS for energy and TCA cycle enzymes are calcium dependent. Further, an optimal range of mitochondrial calcium is crucial for TCA cycle activity as too much or too little mitochondrial calcium will lead to TCA cycle shutdown and cell death. We therefore hypothesize BCL2 regulates mitochondrial calcium levels to ensure they are within the optimal range for TCA cycle activity. Further, we hypothesize venetoclax resistant LSCs have unique calcium biology that can be exploited for therapeutic potential. All studies were done in LSCs isolated from patient samples. In venetoclax sensitive LSCs, inhibition of BCL2 leads to increased mitochondrial calcium content and decreased OXPHOS activity prior to cell death. Inhibition of SERCA, a calcium ATPase responsible for calcium flux into the ER, also leads to increased mitochondrial calcium content and decreased OXPHOS activity prior to cell death. Further, venetoclax resistant LSCs have increased expression of calcium channels responsible for calcium efflux from the ER and influx into the mitochondria. This supports the increased mitochondrial calcium and increased TCA cycle activity seen in venetoclax resistant LSCs. Taken together, our data suggest BCL2 modulates: mitochondrial calcium levels, oxidative metabolism and survival of LSCs. Further, sensitivity to BCL2 inhibition in AML patients is correlated with unique calcium biology properties. Our findings imply venetoclax resistant LSCs may be targeting through transmembrane calcium channels.

Humphrey Petersen-Jones, class of 2017, Thompson lab

The subthalamic nucleus (STN) is routinely targeted for deep brain stimulation (DBS) in the treatment of Parkinson's disease (PD). For treating intractable tremor in PD, several studies report clinical benefit with less side effects when stimulating dorsal to the STN, in the rostral zona incerta (rZI). The rZI is located near three structures involved in motor control: the pallidothalamic tract, cerebellothalamic tract, and the prelemniscal radiation. As such, the rZI is in an ideal position to integrate descending motor signaling with somatosensory feedback. Despite promising results, widespread use has been slow due to the lack of an objective way to verify electrode position in the rZI, leading to inconsistent electrode placement and variability in the stimulated regions. We sought to develop an objective strategy for incorporating intraoperative verification of electrode location within rZI using a combination of neurophysiological and kinesthetic analyses. In seven idiopathic PD patients undergoing awake STN DBS, we measured local field potentials (LFPs) from the rZI, dorsal STN, and ventral STN. Simultaneously, we measured electromyography (EMG) of forearm muscles during three repetitions of four movements: baseline, pronation, supination, and winged. Specifically, we recorded from the flexor carpi radialis, extensor carpi radialis, flexor carpi ulnaris, and extensor carpi ulnaris. We measured the association between oscillatory activity in the brain and muscle group activation using spectral coherence. We hypothesized that differences in LFP spectral power and LFP-EMG coherence would differentiate the rZI, the dorsal STN, and the ventral STN. We found that the extensor ulnaris exhibited muscle group significant coherence in high beta and low gamma frequency bands within rZI compared to both ventral STN and dorsal STN ($p = 0.032$). These preliminary results exhibit differences in LFP-EMG coherence that could potentially be used intraoperatively to verify electrode placement.

Cecilia Levandowski, class of 2012, Taatjes lab

The aging population is rapidly expanding, and with it, the prevalence of chronic diseases such as diabetes, cancer, and Alzheimer's disease. As our understanding of the biology of aging advances, the complexity of the aging process becomes more apparent. The naturally occurring p53 isoform Δ40p53 is expressed in most cell types and is directly implicated in mammalian aging. For example, in mouse models, co-expression of WTp53 and Δ40p53 results in an accelerated aging phenotype with premature development of aging pathologies such as osteoporosis and Alzheimer's disease. The basic mechanisms driving these Δ40p53-dependent cellular and physiological changes remain poorly understood. Human Δ40p53 lacks the first N-terminal transactivation domain of WTp53, and Δ40p53 is preferentially translated during cell stress. The Δ40p53 isoform oligomerizes with WTp53 to form hetero-tetramers with altered function compared to WTp53 tetramers. Co-expression of Δ40p53 and WTp53 results in the formation of a mixed population Δ40p53:WTp53 tetramers, including "contaminating" WTp53 tetramers. This precludes a reliable functional comparison of Δ40p53:WTp53 tetramers vs. WTp53. To circumvent this issue, we developed and validated a strategy—based upon the native p53 tetramer structure—in which Δ40p53 is tethered to WTp53 (Δ40p53:WTp53) as a single transcript, resulting in a pure population of tetramers with a defined 2:2 ratio of Δ40p53 to WTp53. Using CRISPR/Cas9, human cell lines were generated in which Δ40p53:WTp53 (and controls) was inserted at the native TP53 locus. This enabled Δ40p53:WTp53 expression and induction in a physiologically relevant manner. Using these edited cell lines (Δ40p53:WTp53, WTp53, and WTp53:WTp53) in combination with precision run-on nuclear sequencing (PRO-seq), we have been able to determine how the Δ40p53 isoform alters WTp53 transcriptional activity, including changes in the non-coding transcriptome. These results have been complemented with ChIP-seq and other experiments to further define how Δ40p53:WTp53 alters the biological function of WTp53.

Posters

1	Ariel L Hernandez	PARP Inhibition Enhances Radiotherapy of SMAD4 Deficient HNSCCs in Experimental Models
2	Ashley Denney	Selective functional inhibition of tumor-derived p53 mutants by cytosolic chaperones identified via split-YFP in yeast
3	Brian Lloyd	Characterization of neurexin-3-α using vSLENDR
4	Bruce Kirkpatrick	Implementing Covalent Adaptable Networks for Biomedical Applications
5	Connor Hughes	Investigating the role of Eya3 in the regulation of innate immune signaling cascades in Triple Negative Breast Cancer
6	Daniel Youmans	DNA-binding proteins modulate PRC2 occupancy on chromatin and maintain stem cell pluripotency
7	Devin Boe	Tissue-resident alveolar macrophages from young and aged mice respond differently to distal injury
8	Evan Lester	Tau aggregates are RNA-protein assemblies enriched in snRNAs that mis-localize SRSF2 in diverse tauopathies
9	Frances Li	Investigating Mechanisms of Early Viral Clearance by Scavenger Receptor A6 (MARCO)
10	Hans Anderson	Imaging of electrical activity in small diameter fibers of the murine peripheral nerve with virally-delivered GCaMP6f
11	Harry Park	Understanding the mechanism of bone marrow stromal cell mediated protection of FLT3-ITD AML from FLT3 targeted therapy
12	Hei-Yong Lo	Uncovering mechanisms behind RNA localization to the mammalian centrosome
13	Isabel Fernandez	Differential IL-2Rβ organization in memory and naïve CD8+ T cells modulates IL-15 sensitivity
14	Jon Kibbie	The Short Chain Fatty Acid Butyrate Inhibits Pathobiont-Driven Gut CD4 T Cell Activation and HIV-1 Replication.
15	Joseph Hsieh	The Function and Regulation of PODXL in Fusion-Positive Rhabdomyosarcoma

16	Karina Gomez	Cancer cell CD44 mediates macrophage/monocyte-driven regulation of head and neck cancer stem cells.
17	Lily Nguyen	The Consequences of Compromised Mitochondrial Metabolism in Epithelial Cell Differentiation
18	Meagan Chriswell	Mucosal IgA has both Bacterial and Systemic Autoreactivity Targets in Pre-clinical Rheumatoid Arthritis
19	Michael Nash	Maternal western-style diet programs a glycolytic phenotype in fetal non-human primate hematopoietic CD34+ progenitor cells and macrophages, which manifests in chronic liver fibrosis later in life
20	Nathaniel Skillin	Photopolymerized Hydrogels for Expansion Microscopy
21	Rachel Ancar	Coronavirus RNA is cleaved by host and viral endoribonucleases
22	Ricardo Villarreal	The role of intermediate filaments in coordinating group B streptococcus meningitis pathogenesis
23	Robert Jones	Multi-omic interrogation of gemcitabine and cisplatin resistant bladder cancer cell lines identifies unique and shared mediators of chemosensitivity and resistance
24	Ruth Wang	Dynamic regulation of actin-binding protein synaptopodin by butyrate promotes intestinal epithelial barrier function
25	Sarah Haeger	Trusting Your Gut: When a Zealous Bacterium Opens Pandora's Box
26	Sarah Zych	Differential modulation of dopamine and GABA co-release in the dorsal striatum
27	Sean Jones	Role of human APOBEC3 in B Cell Somatic Hypermutation
28	Soraya Shehata	Degrading the C9orf72 Repeat Expansion RNA Foci
29	Taylor Soderborg	The Gut Microbiota in Infants of Obese Mothers Increases Inflammation and Susceptibility to NAFLD
30	Wells LaRiviere	Heparan Sulfate is a substrate for methicillin-resistant S. aureus biofilm formation
31	Will Sheeran	A neuronal signature for monogamous reunion

Excellence in Service Award

This award aims to recognize an outstanding student in the University of Colorado MSTP that improves the lives of their fellow classmates and the experience of the MD/PhD training through their actions. Whether this person serves in an official role (eg: admissions, second look, curriculum committees) or takes initiative in an unofficial capacity (hosts dinners, brings people together across program years), his or her efforts are an example of selfless service.

Congratulations to last year's winner, *Eric Nguyen*!

Acknowledgements and Announcements

Thank you to Matt Becker, Sarah Zych, and all MSTP Student Council members, as well as Grant Lo and the curriculum reform team for their hard work and dedication.

Special thanks to MSTP administration: Arthur, Cara, Patricia, Liz, and Ruhiyah, without whom this event would not be possible.

Thank you to Taylor Soderborg, Brigit High, Taylor Yamamuchi, Sarah Zych, Kelsey Kines, and Bruce Kirkpatrick for organizing the retreat.

Thank you to all MSTP students, faculty, administration, and alumni for their continued participation in and support of the program.

Please join the program in welcoming recruits during revisit weekend:

Friday, March 6th — Sunday, March 8th.

Dinners Friday and Saturday, Denver walking tour Saturday, outdoor activities Sunday.
Contact Dylan, Evan, and Andy for details.

Attendees

Students

Annika Gustafson	MS1
Carley Miller	MS1
Chloe Briney	MS1
Eric Barrientos	MS1
Jordan Hickman	MS1
Keith Dodd	MS1
Mostafa El-Kalliny	MS1
Raquel Ortega	MS1
Uma Kantheti	MS1
Varuna Nangia	MS1
Brenda Seymour	MS2
Bruce Kirkpatrick	MS2
Emily King	MS2
Jackie Turner	MS2
Juan Santiago	MS2
Kelsey Kines	MS2
Nat Skillin	MS2
Thomas Forman	MS2
Will Sheeran	MS2
Amelia Burch	GS1
Brian Lloyd	GS1
Frances Li	GS1
Grant Lo	GS1
Humphrey Petersen-Jones	GS1
Joe Hsieh	GS1
Laurel Darragh	GS1
Lily Nguyen	GS1
Roy Khair	GS1
Soraya Shehata	GS1
Anagha Inguva	GS2
Austin Jolly	GS2
Brigit High	GS2
Connor Hughes	GS2
Meagan Chriswell	GS2
Meghan Kellett	GS2
Michael Nash	GS2
Andy Tekriwal	GS3
Evan Lester	GS3
Faye Camp	GS3
Harry Park	GS3
Leah Bowen	GS3
Marlie Fisher	GS3
Sarah Zych	GS3
Wells LaRiviere	GS3
Daniel Youmans	GS4
Devin Boe	GS4
Elijah Christensen	GS4

Jason Silver	GS4
Karina Gomez	GS4
Rachel Ancar	GS4
Robert Jones	GS4
Ruth Wang	GS4
Ashley Denney	GS5
Isabel Fernandez	GS5
Ari Hernandez	MS3
Ari Hernandez	MS3
Jon Kibbie	MS3
Ricardo Villarreal	MS3
Sean Jones	MS3
Taylor Soderborg	MS3
Tom Vogler	MS3
Cecie Levandowski	MS4
Eric Cross	MS4
Eric Cross	MS4
Greg Kirkpatrick	MS4
Kelly C Higa	MS4
Sarah Haeger	MS4

Faculty and Alumni

Cara Wilson, M.D.
Associate Director, MSTP
University of Colorado

Shawna Cox, Ph.D
Assistant Dean, Graduate School
University of Colorado

Sarah Nelson-Taylor, M.D., Ph.D.
Alumni, '19

Brian L Harry, M.D., Ph.D.
Faculty and Alumni, '16
CU Pathology

Simon Hambidge, M.D., Ph.D
Alumni, '94
University of Colorado, DHHA

Ben Young, M.D., Ph.D.
Alumni, '92
ViiV Healthcare

J. David Port, Ph.D.
Faculty
University of Colorado

Jeff Jacot, Ph.D.
Faculty
University of Colorado

Matthew Taliaferro, Ph.D.
Faculty
University of Colorado

Rajeev Vibhakar, M.D., Ph.D.
Faculty
University of Colorado