Combined Degree Program
Annual Retreat
(Virtual)

August 6 - 7, 2020

Perelman School of Medicine at the University of Pennsylvania
The Combined Degree and Physician Scholar Programs
Administration

Skip Brass, MD, PhD
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Steering Cmt Member
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Steering Cmt Member, VMD-PhD program
Steering Cmt Member, VMD-PhD program
Steering Cmt Member, VMD-PhD program
Steering Cmt Member, VMD-PhD program

Maggie Krall
Nam Narain, PhD
Maura Tucker, MS.Ed
Anouska Bhattacharyya, PhD
David Bittner, MA
Yong No

Director of Administration
Director of Financial Operations
Associate Director, MD-PhD Program
Program Assistant to Skip Brass
Coordinator, MD-PhD Program
Coordinator, VMD-PhD Program
Table of Contents

Welcome Message: Dr. Skip Brass

Welcome Message: Dr. Mike Atchison

Acknowledgements

Agenda

Biographical Sketch of Dr. Tal Zaks

Faculty Talks

Incoming Class

Graduating Student List

Student Talk Abstracts

Poster Session

- Biochemistry & Molecular Biophysics
- Cell and Molecular Biology
- Genetics and Epigenetics
- Microbiology, Virology, and Parasitology
- Genomics & Computational Biology
- Immunology
- Neuroscience
Welcome to the Retreat —  
from the MD-PhD Program Director

I’m writing this on July 31, 2020, 8 months into the COVID-19 pandemic and more than 5 months into the national shutdown required by the pandemic. Lots of things have happened to all of us during that time: Horror at the public health crisis that all of us were supposed to have avoided. Dismay mixed with hope at the overdue national awareness that Black lives matter. Fatigue and stress have become the words of the day, so I am going to use this space to briefly note some of the good things that are happening and that are worth remembering when times are tough.

Over the past 5 months, I’ve heard from many of you. Many of the notes included words of pain, but also words of hope and success, of marriages made and babies born, and of papers published and grants granted. I spent part of my day today reviewing the dean’s letters for the 28 of us who will graduate next May, and preparing to welcome the 36 new family members who will join us this week. The accomplishments of all of our soon-to-be-graduates are a joy to behold and include important new discoveries across the spectrum of biomedical and social sciences.

Each of the 28 has more than fulfilled the hopes that we had when they joined the BMITG™. I suspect that the newest members of the MSTP family will accomplish no less. So, from all of us on the MSTP steering committee to all of you: keep up the good work, take care of yourselves and of each other. Keep making us smile and keep asking for help when you need it.

Huge thanks to all of the members of the retreat planning committee for their care and originality. Finally, a very special welcome to the incoming class. We’re looking forward to seeing you in the real.

Skip Brass, MD PhD  
Director, Penn MSTP
Welcome to the Penn Combined Degree Retreat!

This year certainly has been different. I’m impressed with the resiliency of our students, our faculty, and our institution. As everyone initially scrambled to move to a virtual world, I was amazed at how quickly that occurred, and how we adapted to the situation. But the pandemic is still with us, and we are in a new normal for an indeterminate time. Having a dear friend nearly lose his life to Covid-19 was unnerving. In addition, the events triggered by George Floyd’s murder indicated a more widespread recognition of systemic inequities that have plagued our nation. Having lived through the 1960’s I can only compare this year to 1968 (no history lesson will be provided here). But I am a pathologic optimist. I believe we are experiencing a moment in history when large-scale changes can happen. I believe they will happen (note the pathologic optimism). I am wishing you the best as we ponder the past year, and plan for the future.

Students graduating this year include Amanda Samuels and Elinor Willis, with Robyn Allen scheduled to graduate in December. We are incredibly proud of their accomplishments. Atypically, we have a single student entering this year, Alexander Post. We welcome him to the program and congratulate those graduating now, or in the near future.

I hope you enjoy this day as we experience our first ever virtual retreat. This is the largest MSTP program in the nation, and the only one that includes veterinary combined degree students. I hope you benefit from and appreciate the advantages of the large critical mass and diversity that our program enjoys.

Again, I welcome all current and incoming students.

Sincerely,

Michael Atchison
Many, Many Thanks To the Retreat Planning Committee

We asked the third and fourth year MD-PhD students to take responsibility for planning this event. They did a fabulous job, and we’d especially like to thank the students who were most active in attending the meetings and organizing.

MD-PhDs

John Bernabei
Ryan Boe
Diego Espinoza
Nik Evitt
Jordan Harris
Naveen Jain
Karun Kiani
Jessica Lam
Joyce Liu
Andy Revell
Stacy Thomas
Ellen White
Daniel Xu
## 2020 Incoming Class

### MD/PhD

<table>
<thead>
<tr>
<th>Name</th>
<th>Major</th>
<th>School</th>
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<tbody>
<tr>
<td>Eda Algur</td>
<td>Health Care Management</td>
<td>Harvard</td>
</tr>
<tr>
<td>Masha Alibekova</td>
<td>Bioengineering</td>
<td>CO School of Mines</td>
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<tr>
<td>Vinay Ayyappan</td>
<td>Bioengineering</td>
<td>Hopkins</td>
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<tr>
<td>Carl Bannerman</td>
<td>Cell and Molecular Biology</td>
<td>UMBC</td>
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<td>Sam Chauvin</td>
<td>Immunology</td>
<td>Cornell</td>
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<td>Joy Chiu</td>
<td>Immunology</td>
<td>Yale</td>
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<td>Royce Dong</td>
<td>Bioengineering</td>
<td>Wash U</td>
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<tr>
<td>Sam Dubensky</td>
<td>Immunology</td>
<td>U Chicago</td>
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<tr>
<td>Andres Fernandez del Castillo</td>
<td>Biochemistry &amp; Molecular Biophysics</td>
<td>Notre Dame</td>
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<td>Max Frankfurter</td>
<td>Cell and Molecular Biology</td>
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<td>Eli Gonzalez</td>
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<td>Rob Hapke</td>
<td>Cell and Molecular Biology</td>
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<td>Claudia Heymach</td>
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<td>Blake Jardin</td>
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<td>Paul Kaminski</td>
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<td>Nipun Kottage</td>
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<td>Rachit Kumar</td>
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<td>Maria Merolle</td>
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<tr>
<td>Jeremy Morrissette</td>
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<td>Emmanuel</td>
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<td>Lance Murphy</td>
<td>Bioengineering</td>
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## 2020 Incoming Class

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<th>Name</th>
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<tr>
<td>Sweta Narayan</td>
<td>Cell and Molecular Biology</td>
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<td>Margo Orlin</td>
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<td>Jesse Pace</td>
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<td>Maggie Pecsok</td>
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<td>Jonathan Pham</td>
<td>Pharmacology</td>
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<td>Kenneth Pham</td>
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<td>Carson Poltorack</td>
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<td>Ankita Reddy</td>
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<td>Han-Seul Ryu</td>
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<td>Yusha Sun</td>
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<td>Jonathan Sussman</td>
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<td>Vickie Wang</td>
<td>Neuroscience</td>
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<tr>
<td>Caroline Wechsler</td>
<td>History and Sociology of Science</td>
<td>Harvard</td>
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<tr>
<td>Daniel Yen</td>
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<td>USC</td>
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<tr>
<td>David Zhang</td>
<td>Genomics and Computational Biology</td>
<td>Cornell</td>
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**VMD/PhD**

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<tr>
<th>Name</th>
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<tbody>
<tr>
<td>Alexander Post</td>
<td>Cell and Molecular Biology</td>
<td>Queens U</td>
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</tbody>
</table>
MD/PhDs

Divyansh Agarwal
Multivariate Statistical Analysis in Single Cell Transcriptomics
Advisor: Dr. Nancy R. Zhang

Opeyemi Alabi
Striatal Computations in Health and Psychiatric Disease
Advisor: Dr. Marc Fuccillo

Ahmed Aly
Image Analysis and Shape Modeling for Mitral Valve Surgery
Thesis Advisors: Dr. Paul A. Yushkevich and Dr. Robert C. Gorman

Samuel Belfer
A *Drosophila* Model of Sleep Restriction Therapy for Insomnia and Neurodegenerative Disease
Thesis Advisor: Dr. Matthew Kayser

Dana Bellissimo
The regulation of myeloid inflammatory responses by RUNX1: roles in normal and malignant hematopoiesis
Thesis Advisors: Dr. Nancy Speck and Dr. Gary Gilliland

Leilani Chirino
Negative Regulation of NK Cell Activation by Cbl-b and TAM Receptors
Thesis Advisor: Dr. Taku Kambayashi

Nabil Darwich
Structure and Function Of A Disease Associated Tau Disaggregase
Thesis Advisor: Dr. Edward B. Lee

Amy Davis
The effect of early life exposure on influenza antibody repertoire and subsequent consequences for viral evolution
Thesis Advisor: Dr. Scott E. Hensley

Samir Devalaraja
Tumor-Derived Retinoic Acid Regulates Intratumoral Monocyte Differentiation to Promote Immune Suppression
Thesis Advisors: Dr. Malay Haldar and Dr. George Cotsarelis
Leela Chakravarti Dilley
Genetic control of sleep ontogeny in Drosophila
Thesis Advisor: Dr. Matthew Kayser

Robert Dilley
Mechanisms of telomere repair synthesis
Thesis Advisor: Dr. Roger Greenberg

Melody Esmaeili
Chromatin accessibility and histone acetylation in the regulation of competence in early development
Thesis Advisor: Dr. Peter Klein

Natania Field
A Tale of Two Ligases: Itch and Cul5 limit T cell-mediated inflammatory disease
Thesis Advisor: Dr. Paula Oliver

Joshua Franklin
Following the Child's Lead: Care and Transformation in a Pediatric Gender Clinic
Thesis Advisor: Dr. Adriana Petryna

Piotr Kopinski
Regulation of nuclear epigenome by mitochondrial DNA heteroplasmy.
Thesis Advisor: Dr. Douglas C Wallace

Ian Mellis
Systems Biology of Gene Regulation Across Scales: From Single Molecules to Cellular Identities
Thesis Advisor: Dr. Arjun Raj

Michelle Munyikwa
Up from the Dirt: Racializing Refuge, Rupture, and Repair in Philadelphia
Thesis Advisor: Dr. Deborah Thomas

Andrew Murphy
Informing Neuromodulation Therapies with a Control-Theory Approach to Brain Network Plasticity
Thesis Advisor: Dr. Dani Bassett

Ben Philipson
From Bench to Bedside and Back Again: Car T Cell Signaling and Survival
Thesis Advisors: Dr. Mike Milone and Dr. Steve Albelda
M. Elle Saine
Measuring Disease-Related Stigma among Patients with Chronic Hepatitis C Virus Infection
Thesis Advisor: Dr. Vincent Lo Re, III

Ethan Solomon
Characterization and Perturbation of Functional Networks that Support Human Memory
Thesis Advisor: Dr. Michael Kahana

Katherine Szigety
HDAC3 ensures stepwise epidermal stratification via NCoR/SMRT-reliant mechanisms independent of its histone deacetylase activity
Thesis Advisor: Dr. Sarah E. Millar

Eduardo Torre
Quantitative Characterization and regulation of single cell non-genetic variability in melanoma
Thesis Advisor: Dr. Arjun Raj

Hejia Henry Wang
Cytosolic delivery of inhibitory antibodies with cationic lipids
Thesis Advisor: Dr. Andrew Tsourkas

Michael Werner
Comparative structure-function analysis of BET proteins in transcription
Thesis Advisor: Dr. Gerd Blobel

Krzysztof Wojtak
Preventive and Therapeutic DNA Technologies Targeting Epstein-Barr Virus
Thesis Advisor: Dr. David B. Weiner

Cedric Huchuan Xia
Linking Dimensions of Psychopathology to Functional Brain Networks and Beyond
Thesis Advisors: Dr. Ted Satterthwaite and Dr. Danielle Bassett

Joseph Lee Young
Impossible Terrain: An Ethnography of Policing in Atlantic City, New Jersey
Thesis Advisors: Dr. Adriana Petryna and Dr. Deborah Thomas
2020 MSTP Virtual Retreat

Agenda

Please note – all sessions will be hosted within the same link, unless otherwise noted and times are listed in Eastern Daylight Time (EDT)

Day 1 – August 6, 2020

Opening Remarks
11:00 – 11:15am

Keynote Talk
11:15 – 12:15pm

Dr. Tal Zaks
Chief Medical Officer, Moderna Inc.

Developing an mRNA vaccine for the prevention of COVID-19

Student Talks
12:15 – 1:15pm

Aaron Williams
Sound improves neural encoding of stimulus direction in mouse V1.

Jing Luan
Distinct properties and functions of CTCF revealed by a rapidly inducible degradation system

Break
1:15 – 2:00pm

Faculty Talks
2:00 – 2:45pm

Jennifer Orthmann-Murphy, MD, PhD
An Undiagnosed White Matter Disorders Clinic / Seventeen years of physician scientist training led me right back to my graduate thesis

Riccardo Gottardi, PhD
Establishing a pediatric regenerative medicine and drug delivery lab / how the crosstalk between research and LGBTQ+ activism helped me in my career

Elizabeth J. Bhoj, MD, PhD
Pediatric Genetic Diseases: How do we get every patient a diagnosis and targeted treatment?

Student Poster Session via YouTube and Slack*
2:45 – 4:00pm
2020 MSTP Virtual Retreat

Agenda

*Please note – all sessions will be hosted within the same link, unless otherwise noted and times are listed in Eastern Daylight Time (EDT)

Day 2 – August 7, 2020

Opening Remarks
11:00 – 11:15am

Student Talks
11:15 – 12:15pm

Aileen Ren
*Downstream mechanisms of vascular malformation*

Joe Park
*Exome-wide evaluation of rare coding variants using electronic health record data to identify novel gene-phenotype associations*

Break
12:15 – 1:00pm

Social Activity
1:00 – 2:00pm

Alumni Panel*
2:00 – 3:00pm
(passcodes were emailed ahead of time to “pre-registrants,” if anyone wants a passcode for a different panel that they didn’t sign up for in advance, please email David or Maura)

*Careers in Academia - Fellowship/Instructorship*

Dania Daye
Andrew Stern
Jessica Shay
Theodore Drivas
David Hill

*Careers in Industry*

Michael Detke
Peter Hammerman
Tamara Wexler
David Margolin
Andrew Trister
Careers in the Social Sciences
Sydney Brown
Marta Rowh
Daniel Wollman
Carla Keims

Careers as Surgeon-Scientist
Peter Gruber
Jason Wertheim
Nicholas Parrish

Careers in Academia- Professorship
Stuart Lipton
Marcela Maus
Ralph DeBerardinis
Thao Nguyen
Tamar Gur

*additional links and/or passcodes will be provided for these sessions
As Chief Medical Officer, Tal Zaks oversees clinical development and regulatory affairs across Moderna. Prior to joining Moderna, Dr. Zaks was senior vice president and head of Global Oncology at Sanofi, where he was responsible for all aspects of oncology drug discovery, development and commercialization.

Dr. Zaks began his industry career at GlaxoSmithKline in the genetics research group, where he built the oncology translational medicine team and led translational research on lapatinib as well as the in-licensing and clinical development of foretinib. In addition to his industry work, Dr. Zaks is associate professor of medicine at the University of Pennsylvania, and has served as a volunteer physician at the Philadelphia Veterans Administration Medical Center, treating patients with genitourinary cancers.

Dr. Zaks received his M.D. and Ph.D. from the Ben Gurion University in Israel and conducted post-doctoral research at the U.S. National Institutes of Health. He completed his clinical training in internal medicine at Temple University Hospital followed by a fellowship in medical oncology at the University of Pennsylvania.

Dr. Zaks serves on the Board of Directors of Adaptimmune Therapeutics plc.
Dr. Jennifer Orthmann-Murphy, MD, PhD, is an Attending Physician and Assistant Professor of Neurology at Penn. She earned her BA in the Biological Basis of Behavior from Penn in 2001, and graduated from the MD/PhD program at Penn in 2010. She completed her residency in Neurology at Penn in 2014 and was a Clinical Neuroimmunology and Postdoctoral Research Fellow and postdoctoral fellow at John’s Hopkins School of Medicine from 2014-2018. Currently Co-Director of Age Span Fellowship in MS/Neuroinflammatory Disorders at Penn, Dr. Orthmann-Murphy will speak about her experience creating a new clinic and her research on the dynamic role of glial cells in acquired and inherited demyelinating disease.

Dr. Riccardo Gottardi, PhD, earned his BSc in Applied Physics from the University of Pisa in 2003, and earned his PhD from the University of Genova in 2007. In 2011 he moved to the University of Pittsburgh thanks to a fellowship from the Ri.MED Foundation, and in 2017 he became research assistant professor in the Department of Orthopaedic Surgery of the University of Pittsburgh. Since 2019, Dr. Gottardi has led CHOP’s Bioengineering and Biomaterials laboratory. Dr. Gottardi will speak about his research and career path, as well as his activism through advocacy groups and NGOs campaigning for LGBTQ+ rights.

Dr. Elizabeth J. Bhoj, MD, PhD, is an Attending Physician and Assistant Professor of Pediatrics at CHOP. She earned her BS in Biology/Philosophy from the College of New Jersey in 2002 and graduated from the MD/PhD program at UT Southwestern in 2010. She completed a combined residency in Pediatrics and Medical Genetics at CHOP and stayed to complete a fellowship in Molecular Genetics in 2016, simultaneously earning a Master’s in Translational Research from Penn. The recent winner of a Career Award for Medical Scientists from the Burroughs Wellcome Foundation, Dr. Bhoj is now in her third year of being a PI and will speak about her career trajectory, research on targeted treatment of human genetic disorders, and her work promoting clinical, research, and translational efforts in under-resourced countries.
Student Talks

Aaron Williams
*Sound improves neural encoding of stimulus direction in mouse V1.*
Advisor: Dr. Maria Geffen

Jing Luan
*Distinct properties and functions of CTCF revealed by a rapidly inducible degradation system*
Advisor: Dr. Gerd Blobel

Aileen Ren
*Downstream mechanisms of vascular malformation*
Advisor: Dr. Mark Kahn

Joseph Park
*Exome-wide evaluation of rare coding variants using electronic health record data to identify novel gene-phenotype associations*
Advisors: Dr. Daniel J Rader and Dr. Marylyn D Ritchie
Sound improves neural encoding of stimulus direction in mouse V1

Aaron Williams, Dr. Maria Geffen

Submitted by: Aaron Williams, Neuroscience
Email: aaron.williams@pennmedicine.upenn.edu
Advisor: Dr. Maria Geffen

In the natural world, we integrate visual and auditory signals during behaviors such as navigation and communication. Auditory and visual inputs can modulate the perception of the complementary modality, but the neural correlates of audiovisual integration are not fully understood. In the visual cortex, auditory stimuli modulate light-evoked firing rates of individual neurons. Here, we investigated how auditory stimuli modulate other aspects of neural processing in addition to firing rate, and whether this results in improved neural encoding of the visual stimulus. We presented visual drifting gratings with and without simultaneous auditory white noise to awake mice while recording neuronal activity in the primary visual cortex (V1). Sound modulated the light-evoked activity of 70% of light-responsive neurons, the majority of which increased their activity in association with sound. These firing rate changes were accompanied by increased response duration and reduced response latency. Additionally, across contrast levels sound reduced the variability of the light-evoked response, shown by a reduction in the response coefficient of variation. In individual neurons that were additionally direction-selective, we found that sound improved the discriminability of the preferred direction over all other directions. Furthermore, sound improved the neural population’s encoding of the drifting grating direction. These improvements in neural encoding were greatest at low to intermediate contrast levels. These results demonstrate that simultaneous auditory input enhances the light-evoked response magnitude and timing and decreases variability in individual neurons, resulting in improved stimulus encoding at the individual neuron and population level. These findings expand our knowledge of how multisensory processing is mediated at a neural level, and provides a foundation for improved neural-based auditory and communication assistive devices.
Distinct properties and functions of CTCF revealed by a rapidly inducible degron system

Jing Luan, Guanjue Xiang, Pablo A. Gómez-García, Jacob M. Tome, Zhe Zhang, Marit W. Vermunt, Haoyue Zhang, Anran Huang, Cheryl A. Keller, Belinda M. Giardine, Yu Zhang, Yemin Lan, John T. Lis, Melike Lakadamyali, Ross C. Hardison, and Gerd A. Blobel

Submitted by: Jing Luan, CAMB - Genetics and Epigenetics
Email: jluan@pennmedicine.upenn.edu
Advisor: Dr. Gerd Blobel

CCCTC-binding factor (CTCF) is a conserved zinc finger transcription factor implicated in a wide range of functions, including genome organization, transcription activation and elongation. To explore the basis for CTCF functional diversity, we coupled an auxin-induced degron (AID) system with precision nuclear run-on (PRO-seq). Unexpectedly, oriented CTCF motifs in gene bodies are associated with transcriptional stalling in a manner ostensibly independent of bound CTCF. Moreover, CTCF at different binding sites (CBSs) display highly variable resistance to degradation. Motif sequence does not predict degradation behavior, but location at chromatin boundaries and chromatin loop anchors, as well as co-occupancy with cohesin are associated with delayed degradation. Single-molecule tracking experiments link CTCF degradation resistance to chromatin residence time, which has ramifications regarding architectural CTCF functions. Our study highlights the heterogeneity of CBSs and uncovers intrinsic properties specific to topologically important CBSs, thus providing insights into the basic processes of genome organization and transcription regulation.
Cerebral cavernous malformations (CCMs) are vascular malformations comprised of clusters of dilated capillaries that develop in the central nervous system and are associated with high morbidity such as seizures, neurologic deficits, and hemorrhagic stroke. CCMs are found in 0.5% of the population worldwide and have two forms, familial and sporadic. Familial forms of CCM disease are caused by loss-of-function mutations in the genes CCM1 (krit1), CCM2, or CCM3 (pdcd10), together which make up the CCM adaptor protein complex. The neonatal mouse model for studying this disease has been instrumental in elucidating downstream signaling pathways. When the CCM genes are deleted in neonatal mouse endothelium at P1 (1st day of life), the mice develop numerous hindbrain lesions by P10. However, a puzzling observation known to the field is that deletion of the CCM genes after this crucial neonatal window does not produce lesions. This phenotype is in contrast to the fact that humans can develop CCM lesions throughout their lifetime.

Studies in the vascular malformation field have implicated mutations in the PI3K pathway important in angiogenesis, in the formation of venous and lymphatic malformations. My project proposes that 1) the neonatal model harbors a permissive environment for development of lesions which the adult brain vasculature lacks, 2) activating mutations in pro-angiogenic genes can permit lesion formation in adult animals, and 3) human lesions display such synergy. Our studies reveal a synergistic interaction between CCM loss and pro-angiogenic signaling pathways that underlie a significant proportion of human CCM lesions. These findings contribute to better understanding of disease pathogenesis and provide novel therapeutic targets for CCM disease.
Exome-wide evaluation of rare coding variants using electronic health record data to identify novel gene-phenotype associations


Submitted by: Joseph Park, Genomics and Computational Biology
Email: joseph.park@pennmedicine.upenn.edu
Advisors: Dr. Daniel J Rader and Dr. Marylyn D Ritchie

The clinical impact of rare loss-of-function variants in most genes has yet to be determined. Integrating DNA sequencing with electronic health records (EHR) could enhance our understanding of the contribution of rare genetic variation to human disease. Leveraging 10,900 whole exomes linked to EHR data in the Penn Medicine Biobank (PMBB) for discovery, we addressed the association of the cumulative effect of rare predicted loss-of-function (pLOF) variants per gene on an exome-wide scale with a phenome of diverse EHR phenotypes. After discovering 97 exome-by-phenome-wide significant gene-disease associations (p < 10^{-6}), we robustly replicated 26 of these in PMBB, three other ‘medical’ biobanks, and the population-based UK Biobank (UKB). Five gene-disease associations represented ‘positive controls’ and 21 were novel findings. We show the value of aggregating rare pLOF variants into ‘gene burdens’ for association with EHR phenotypes in a medical biobank to identify novel clinical relationships for mutated human genes. We suggest that this approach applied to even larger numbers of individuals will yield many new insights into the relationship between rare genetic variation and disease phenotypes.
For the virtual poster presentation, please join our Penn 2020 MSTP Retreat Slack. When you join, you will be able to see a channel for each of the posters included in this year’s MSTP retreat. The video presentation for each poster will available via the links below and in the individual Slack channels where you can discuss or ask questions about each poster. If you have any questions, there is a welcome-posters channel that can help!

**Biochemistry & Molecular Biophysics**

Poster 1  
**Abstract** - Presentation  
*Discovery of an unnatural DNA modification derived from a natural secondary metabolite*  
Presenter: Tong Wang | Advisor: Dr. Rahul Kohli

**Cell and Molecular Biology**

**Genetics and Epigenetics**

Poster 2  
**Abstract** - Presentation  
*Investigating the Effect of Insulin on BCAA Oxidation in Health and Disease*  
Presenter: Marc Bornstein | Advisor: Dr. Zoltan Arany

Poster 3  
**Abstract** - Presentation  
*High-performance CRISPR-Cas12a genome editing for combinatorial genetic screening*  
Presenter: Niklaus Evitt | Advisors: Dr. Rahul Kohli and Dr. Junwei Shi

**Microbiology, Virology and Parasitology**

Poster 4  
**Abstract** - Presentation  
*Leveraging the human skin commensal Alcaligenes faecalis to improve early wound healing*  
Presenter: Ellen White | Advisor: Dr. Elizabeth Grice

Poster 5  
**Abstract** - Presentation  
*Cellular Roles and Mechanisms of IL-1 Signaling During Control of Salmonella Typhimurium Infection*  
Presenter: Jenna Zhang | Advisors: Dr. Igor Brodsky and Dr. Sunny Shin

**Genomics & Computational Biology**

Poster 6  
**Abstract** - Presentation  
*Role of YY1 in chromatin looping throughout the cell cycle*  
Presenter: Jessica Lam | Advisor: Dr. Gerd Blobel
Immunology

Poster 7
**Abstract** - **Presentation**
*Interrogating immune signatures in the thoracic duct of patients with multiple sclerosis*
Presenter: Diego Espinoza | Advisor: Dr. Amit Bar-Or

Poster 8
**Abstract** - **Presentation**
*The role of skin microbiota in TSLP-mediated skin barrier function*
Presenter: Jordan Harris | Advisors: Dr. Elizabeth Grice and Dr. Taku Kambayashi

Neuroscience

Poster 9
**Abstract** - **Presentation**
*Altered functional brain dynamics in chromosome 22q11.2 deletion syndrome during facial affect processing*
Presenter: Eli Cornblath | Advisor: Dr. Danielle S. Bassett

Poster 10
**Abstract** - **Presentation**
*Deficits in axonal autophagosome transport caused by a mutation linked to Parkinson’s disease*
Presenter: Dan Dou | Advisor: Dr. Erika Holzbaur
Poster 1 | Biochemistry & Molecular Biophysics

discovery of an unnatural DNA modification derived from a natural secondary metabolite

Tong Wang and Rahul Kohli

Submitted by: Tong Wang, Biochemistry & Molecular Biophysics
Email: Tong.Wang@pennmedicine.upenn.edu
Advisor: Dr. Rahul Kohli

Despite widespread interest for understanding how modified bases have evolved their contemporary functions, limited experimental evidence exists for measuring how close an organism is to accidentally creating a new, modified base within the framework of its existing genome. Here, we describe the biochemical and structural basis for how a single point mutation in E. coli’s naturally occurring cytosine methyltransferase can surprisingly endow a neomorphic ability to create an unnatural DNA base, in vivo. Mass spectrometry, bacterial genetics, and structure-guided biochemistry reveal this base to be exclusively derived from a natural but sparse secondary metabolite. Our discovery of a new, unnatural DNA modification reveals insights into the substrate selectivity of DNA methyltransferase enzymes, offers a promising new biotechnological tool for the characterization of the mammalian epigenome, and provides an unprecedented model for how neomorphic bases could arise in nature from repurposed host metabolites.
Type 2 diabetes is a major cause of morbidity and mortality in the U.S. and around the world, and its prevalence is only expected to rise in the coming decades. The key hallmark of the disease is insulin resistance (IR), the increased requirement for insulin in order to take up glucose from the blood. Epidemiological studies have shown that IR is tightly associated with elevated plasma levels of branched chain amino acids (BCAAs: leucine, isoleucine, and valine). Recent studies suggest that elevated BCAAs contribute causally to IR, and genetic data simultaneously suggest that IR contributes to elevated BCAAs, forming a potential positive feedback loop. Pharmacologic interruption of this feedback loop may thus provide a novel therapeutic target to treat IR and diabetes. Interestingly, insulin itself has been shown to promote catabolism of BCAAs in both humans and rodents. However, it remains unknown how insulin promotes oxidation of BCAAs, how this effect may be dysregulated in the setting of insulin resistance, and whether any such dysregulation contributes further to IR. The rate-limiting enzyme of BCAA oxidation is the BCKDH complex. BCKDH activity is inhibited by specific phosphorylation at S293 of the BCKDHA subunit. Preliminary results from our lab now suggest that insulin promotes rapid dephosphorylation of BCKDH in cell culture, and, further, that this response is dependent on mTORC1, an important signaling complex downstream of insulin signaling which coordinates a broad array of metabolic processes. Based on these findings, I hypothesize (1) that insulin promotes BCAA oxidation in a cell-autonomous manner by dephosphorylating BCKDHA S293 through mTORC1-dependent activation of the BCKDHA phosphatase PPM1K; and (2) that in the setting of IR, insulin-induced BCKDHA dephosphorylation is impaired, which contributes to elevated plasma BCAAs and further promotes IR through a positive feedback loop. To address this hypothesis, I will utilize isotope tracing experiments combined with genetic and pharmacological interventions to elucidate the mechanism for insulin-induced BCAA oxidation. Additionally, I will use a hyperinsulinemic-euglycemic clamp to test whether insulin-induced BCKDHA dephosphorylation is recapitulated in vivo, and whether this response is impaired in insulin-resistant mice; further, I will challenge BCKDK knock-out mice, which lack phosphorylated BCKDHA in all tissues, to a high-fat diet to test whether ablation of BCKDHA phosphorylation protects against IR. Together, these experiments will examine the possibility of a positive feedback loop between insulin resistance and elevated BCAA levels, and will test key biochemical elements that may be involved. The insights generated through this proposal will therefore be critical for future pharmacological efforts targeting BCAA metabolism for the treatment of insulin resistance and type 2 diabetes.
CRISPR-based genetic screening has revolutionized cancer drug target discovery, yet reliable, multiplex gene editing to reveal synergies between gene targets remains a major challenge. Here, we present a simple and robust CRISPR-Cas12a-based approach for combinatorial genetic screening in cancer cells. By engineering the CRISPR-AsCas12a system with key modifications to the Cas protein and its CRISPR RNA (crRNA), we can achieve high efficiency combinatorial genetic screening. We demonstrate the performance of our optimized AsCas12a (opAsCas12a) through double knockout screening against epigenetic regulators. This screen reveals synthetic sick interactions between Brd9&Jmj6, Kat6a&Jmj6, and Brpf1&Jmj6 in leukemia cells.

*Adapted from *Nat Commun* 11, 3455 (2020). [https://doi.org/10.1038/s41467-020-17209-1](https://doi.org/10.1038/s41467-020-17209-1). To view a copy of the license, visit [https://creativecommons.org/licenses/by/4.0/](https://creativecommons.org/licenses/by/4.0/).
Non-healing wounds present a major challenge to the healthcare system, as they are responsible for significant treatment costs as well as high rates of morbidity and mortality. Standard of care treatments often fail to heal chronic wounds, underscoring the need to identify new therapeutic approaches. One source of novel therapeutic targets is the skin microbiome, which exists at the interface of cutaneous wounds. Our lab has previously shown that the colonizing microbiota of diabetic foot ulcers (DFUs) influences healing outcomes. One such microbe is *Alcaligenes faecalis*, which was previously dismissed as an environmental bacterium but was surprisingly prevalent and abundant in DFU wound samples. Treatment of wounds with *A. faecalis* lead to accelerated wound closure as well as increased keratinocyte migration and proliferation. Pro-inflammatory cytokine signaling in keratinocytes is necessary to coordinate this wound healing response, and IL-6 in particular has been shown to induce keratinocyte migration and proliferation. Therefore, I tested if *A. faecalis* induced a cytokine response in keratinocytes, and found robust IL-6 production after treatment with *A. faecalis* conditioned media. A primary mechanism by which bacteria can modulate host responses is through production of small molecules. A genetic approach can be used to identify such bioactive small molecules because they are typically products of distinct genomic regions known as biosynthetic gene clusters (BGCs). I mined the whole genome sequence of the *A. faecalis* isolate that accelerated wound closure for these BGCs. I identified two candidate clusters that encode for molecules that have been shown to modulate keratinocyte behavior. Together, these findings lead to my hypothesis that *A. faecalis* produces BGC-encoded small molecules that improve early wound healing responses by inducing IL-6 production in keratinocytes. In Aim 1, I will determine if *A. faecalis*-mediated IL-6 production by keratinocytes is necessary for the pro-healing response both *in vitro* and *in vivo*. In Aim 2, I will identify the *A. faecalis* BGC responsible for the pro-wound healing phenotype. This study will identify how a commensal microbe can improve the wound healing response, which can serve as a foundation for development of skin microbiota-derived therapies.
Enteric pathogens are a major public health threat, infecting over 1.7 billion people annually and causing diarrheal illness that can progress to dehydration, sepsis and death. Following oral acquisition, enteric bacterial pathogens colonize the human gastrointestinal (GI) tract, where they confront a complex landscape of host defense mechanisms. A diverse array of intestinal cell types mount cell-intrinsic inflammatory responses against bacteria, communicating signaling molecules to coordinate antimicrobial efforts. Uncovering these signaling pathways is critical to understand how hosts subvert bacterial pathogenesis and to identify potential immunomodulatory antibacterial therapies in the face of rising antibiotic resistance rates. Among enteric bacteria, *Salmonella enterica* serovar Typhimurium (*S.* Typhimurium or *S* *T*m) is a leading cause of human GI disease. *S.* Typhimurium invades multiple cell types, including macrophages and intestinal epithelial cells (IECs) which can mount cell-intrinsic inflammatory responses against bacteria. Upon invasion, bacterial components are sensed by innate immune components, leading to a proinflammatory form of cell death known as pyroptosis and processing and release of inflammatory molecules, including the IL-1 family cytokines. IL-1 family cytokines, of which IL-1α and IL-1β are members, induce a variety of downstream effects including innate immune cell recruitment, cytokine production, angiogenesis and immune homeostasis. IL-1 is important in control of *S* *T*m infection in vivo as *Il1r1−/−* mice demonstrate increased mortality and systemic bacterial burdens as compared to wild type mice. However, a key gap in our knowledge is understanding how IL-1-mediated responses enable control of *S* Typhimurium infection. Therefore, the central goal of this study is to define the key cell types involved in producing and responding to IL-1. Macrophages are a major intracellular niche for *S* *T*m, and infection in vitro leads to abundant IL-1 production. In contrast, infected intestinal epithelial cells do not produce large amounts of IL-1, but express *Il1r1* and upregulate antimicrobial defenses in response to IL-1 in other infectious contexts in vitro. I hypothesize that IL-1 released from intestinal macrophages signals to intestinal epithelial cells to restrict *S.* Typhimurium infection. These studies will further our understanding of the cellular mechanisms underlying IL-1-mediated control of *S.* Typhimurium infections, providing the groundwork for future mechanistic studies of cytokine responses and potential immunomodulatory targets to treat infection in humans.
Mitosis is marked by a global cessation of transcription, eviction of transcription factors, and the dissolution of most chromatin structure. During the mitosis to G1 phase transition, cells must therefore address the challenge of rapidly re-establishing previous 3D genome organization. However, much remains unknown about how cells transition from a relatively disorganized chromatin state to a cell type-specific conformation. While CTCF and cohesin-mediated loop extrusion has been shown to forge some chromatin loops, many observed architectural features cannot be explained by this mechanism. Another important architectural factor, YY1, has been implicated in enhancer-promoter loops in studies in interphase cells. However, its dynamics and role in chromatin loop formation has not been explored at the critical juncture between mitosis and G1 phase. We hypothesize that YY1 is required for forming chromatin loops upon exit from mitosis. We aim to characterize YY1 occupancy as it relates to the emergence of chromatin loops and then test its necessity by interrogating effects of global depletion during mitosis. We also propose studying the mechanisms of YY1-mediated loop formation by predictive modeling of loops based on local genetic and epigenetic context. Finally, we will test the sufficiency of YY1 and its different functional domains by performing forced looping studies. By studying YY1 during the mitosis to G1 phase transition and investigating the requirements for loop formation, we hope to gain new insights into the fundamental mechanisms underlying genome organization.
Interrogating immune signatures in the thoracic duct of patients with multiple sclerosis

Diego A. Espinoza, Rui Li, Amit Bar-Or

Submitted by: Diego A. Espinoza, Immunology
Email: diego.espinoza@pennmedicine.upenn.edu
Advisor: Dr. Amit Bar-Or

Multiple sclerosis (MS) is an immune-mediated demyelinating and neurodegenerative chronic disease of the central nervous system (CNS). While a number of immune pathways have thus far been implicated in the immunopathophysiology of MS, a complete understanding has remained elusive. One of the most significant barriers in understanding MS immunopathophysiology is the limited access to two particular MS-relevant immune compartments: the CNS and lymphoid tissues (LTs). A novel approach that would address the shortcomings of existing methods in accessing these tissues is to instead access lymphocytes from the human deep efferent lymphatics (DELS). The DELs, which collect in the thoracic duct (TD), are the drainage point for numerous LTs in the body and part of the CNS. As such, the DELs represent a rich periscope into immune compartments critical to MS immunopathophysiology. To this end, our laboratory has established a collaboration with the Hospital of the University of Pennsylvania by which we cannulate and sample lymphocytes from the human TD. I herein propose a first-of-its-kind interrogation of the human TD immune compartment in patients with MS to find previously poorly characterized and/or unidentified immune signatures of MS. As much is still unknown about the immune phenotypes and networks implicated in MS, a relatively unbiased approach is necessary. Thus, I will leverage a single-cell multiomics approach (CITE-seq) in order to best provide insight into disease immunopathophysiology. I hypothesize that this deep unbiased characterization of TD lymphocytes will provide an opportunity to identify novel immune signatures implicated in MS immunopathophysiology. To test this hypothesis, I will first identify the phenotypic and transcriptomic dysregulations at a cellular level present in the TD of patients with MS compared with controls. I will then investigate the compartmentalization of these signatures to the TD compared with the PB. Overall, these findings will serve as a basis on which to guide further, targeted studies into the immunopathophysiology of MS.
The skin is a physiologic barrier which acts as a first line of defense against infection by foreign pathogens, utilizing physical, chemical, and immunologic mechanisms to prevent microbial invasion. One protection method employed by the skin is the secretion of sebum: a lipid-rich substance produced by dermal-dwelling sebaceous glands (SGs). Sebum contains fatty acids and induces antimicrobial peptide (AMP) expression which limit skin microbial overgrowth and prevent infection. Sebum secretion increases with puberty onset and is thereafter regulated in part by androgenic hormones. Although sebum has a well-defined immunologic function, it has yet to be established if the immune system regulates sebum secretion. Unexpectedly, our lab found that the keratinocyte derived cytokine Thymic Stromal Lymphopoietin (TSLP) promotes sebum secretion, supporting the existence of an immune-sebum regulatory circuit. Our lab has shown that TSLP promotes sebum secretion, not directly through SGs, but intriguingly through stimulation of activated T cells via the TSLP receptor. Accordingly, TSLP- and T cell-deficient mice display a significant reduction in sebum secretion. This surprising finding suggests a novel paradigm whereby adaptive immune cells possess innate immune functionality by regulating barrier function via sebum. We propose to identify the activating signal that mediates this T cell response and initiates immune-sebum regulation. One signal that could allow for T cell activation is skin-specific microbial antigens. Commensal bacterial communities promote tissue-specific immune system development and are necessary for healthy immune function, including generation of tissue-resident, microbial-specific T cells. It is then possible that the skin microbiome is involved in regulating SG function through T cell activation. Indeed, my preliminary data show that germ-free mice secrete less sebum and display less SG-related gene expression than controls. We have also found that mice overexpressing TSLP show increased AMP expression and sebum secretion. I hypothesize that the skin microbiome induces TSLP-mediated, microbial-specific T cell-dependent sebum secretion, promoting skin barrier function and acting as an important homeostatic innate defense against skin infection. To test this, I will (1) establish if the microbiome regulates sebum secretion through formation of microbiota-specific T cells and (2) determine if TSLP-mediated sebum secretion promotes barrier function and infection resistance. Completion of the proposed studies will establish a novel mechanism by which the immune system and skin microbiome coordinate to regulate sebum secretion to promote innate barrier function to identify novel therapeutic targets for common skin conditions associated with dysregulated sebum levels, such as acne vulgaris and atopic dermatitis.
Altered functional brain dynamics in chromosome 22q11.2 deletion syndrome during facial affect processing


Submitted by: Eli Cornblath, Neuroscience
Email: Eli.Cornblath@pennmedicine.upenn.edu
Advisor: Dr. Danielle S. Bassett

Chromosome 22q11.2 deletion syndrome (22q11.2DS) is a multisystem disorder associated with multiple congenital anomalies, variable medical features, and neurodevelopmental differences resulting in diverse psychiatric phenotypes, including marked deficits in facial memory and social cognition. Neuroimaging in patients with 22q11.2DS has revealed differences relative to matched controls in BOLD fMRI activation during facial affect processing tasks, but time-varying interactions between brain areas during facial affect processing have not yet been studied in 22q11.2DS. We applied constrained principal component analysis to identify spatiotemporal brain activation patterns from BOLD fMRI data acquired during an emotion identification task from 58 patients with 22q11.2DS and 58 age-, race-, and sex-matched healthy controls. We measured spatial alignment between these activation patterns and cortical morphometry difference maps from a separate study. Delayed frontal-motor feedback signals were diminished in patients with 22q11.2DS, as were delayed emotional memory signals engaging amygdala, hippocampus, and entorhinal cortex. Task-related engagement of motor and visual cortices and salience-related insular activation were largely unaffected in 22q11.2DS, though insular activation was associated with task performance within the 22q11.2DS sample. Differences in cortical surface area, but not cortical thickness, were slocaloized to an activation pattern associated with face processing. Our findings suggest that relative to matched controls, primary visual processing and insular function is intact in 22q11.22DS patients, while motor feedback, face processing, and emotional memory processes are disrupted. Such insights may help inform potential interventional targets and enhance the specificity of neuroimaging indices of cognitive dysfunction in 22q11.2DS.
**Deficits in axonal autophagosome transport caused by a mutation linked to Parkinson’s disease**

Dan Dou, Alex Boecker, Erika Holzbaur

Submitted by: Dan Dou, Neuroscience
Email: dan.dou@pennmedicine.upenn.edu
Advisor: Dr. Erika Holzbaur

Parkinson’s Disease (PD) is the second-most prevalent neurodegenerative disease and the fastest-growing. Current therapies do not address the underlying progressive loss of neuronal populations, most prominently in the substantia nigra pars compacta (SNc). Leucine-rich repeat kinase 2 (LRRK2) is a leading candidate for a nexus bridging dysregulation of autophagy and PD pathogenesis. Experiments manipulating LRRK2 kinase activity and expression levels have shown changes in levels of autophagy markers, but the specific mechanistic role of LRRK2 in autophagy remains unclear. LRRK2 was recently shown to phosphorylate a subset of Rab GTPases, which are master regulators of membrane trafficking. Mutations in LRRK2 are the most common genetic cause of PD, and LRRK2 kinase activity has also been linked to idiopathic PD. Recently, another PD-associated mutation in VPS35 (a component of the retromer complex) has been reported to increase LRRK2 kinase activity. There is therefore accumulating evidence that there may be multiple PD-causative pathways converging on LRRK2 kinase activity. The goal of this project is to investigate the mechanisms by which LRRK2 dysfunction may contribute to dysregulation of autophagy and axonal transport. Our preliminary data suggests that the most common pathogenic mutation in LRRK2, p.G2019S, causes disruption of autophagosome (AP) transport through increased pausing in multiple model systems, including mouse cortical neurons and human iPSC-derived neurons. In addition, we used MLi-2, a specific kinase inhibitor, to show that this AP pausing phenomenon is dependent on LRRK2 kinase activity. We hypothesize that LRRK2-p.G2019S and VPS35-p.D620N disrupt autophagy through a common mechanism dependent on increased LRRK2 kinase activity and augmented Rab phosphorylation. Future directions for the project are aimed at further elucidating the mechanism of AP pausing and establishing whether these pathogenic mutations cause functional deficits in AP acidification and cargo degradation as a possible mechanism for neurodegeneration. We hope that this work will have broad implications for fundamental improvements to therapies combatting neuronal loss in PD.
Alumni Panel Discussions

Careers in Academia - Fellowship/Instructorship

- Dania Daye (https://www.linkedin.com/in/ddaye)
  - Instructor of Radiology at Mass General Hospital/Harvard Medical School
  - Also willing to discuss: Mentoring, Women Physician-Scientists, American
    Physician Scientists Association
- Andrew Stern (https://connects.catalyst.harvard.edu/Profiles/display/Person/150514#)
  - Fellow in Behavioral Neurology at Brigham and Women’s
- Jessica Shay (https://connects.catalyst.harvard.edu/Profiles/display/Person/172543)
  - Fellow in Medicine at Massachusetts General Hospital
- Theodore Drivas (https://www.chop.edu/doctors/drivas-theodore-g)
  - Fellow in Division of Human Genetics at CHOP
  - Also willing to discuss: Choosing a PhD mentor, Genetics as a clinical field,
    LGBTQ-related topics
- David Hill (https://www.chop.edu/doctors/hill-david-a)
  - Instructor of Pediatrics, Perelman School of Medicine
  - Also willing to discuss: Young family

Careers in Industry

- Michael Detke (https://www.cortexyme.com/team/)
  - Chief Medical Officer at Cortexyme
  - Other notes of interest: Psychiatry MD/PhD with careers in various industry
    settings (large Pharma, small biotech, clinical trial sites)
- Peter Hammerman (https://www.linkedin.com/in/peter-hammerman-50a43854)
  - Global Head, Oncology Translational Research at Novartis
  - Also willing to discuss: Initial faculty position, Early career science, Industry
    transition
- Tamara Wexler (https://www.linkedin.com/in/tamara-wexler-70a9163)
  - Clinical Associate Professor, Department of Rehabilitation Medicine; Director of
    Pituitary Center at NYU Langone Health
  - Managing Director, TWX Consulting
  - Other notes of interest: Formerly in biotech R&D consulting while also attending
    in academic medicine
  - Also willing to discuss: Creating your own path, Children, Mentoring
- David Margolin (https://www.linkedin.com/in/david-margolin-36293671)
  - Senior Vice President, Clinical and Translational Medicine at Cerevance
  - Other notes of interest: Has been in industry for 15 years, previously on
    Neurology staff at MGH for 20 years
- Andrew Trister (https://www.linkedin.com/in/trister)
  - Deputy Director, Bill and Melinda Gates Foundation
  - Other notes of interest: Formerly led a clinical trial and a machine learning group
    at Apple
  - Also willing to discuss: Mentoring
Careers in the Social Sciences

- **Sydney Brown** ([https://www.linkedin.com/in/sydney-brown-b09705a](https://www.linkedin.com/in/sydney-brown-b09705a))
  - Fellow in Pediatric Anesthesiology, Research Fellow at CHOP
  - Other notes of interest: Research in health services, clinical epidemiology
- **Marta Rowh** ([https://www.stcharleshealthcare.org/providers/marta-rowh-md-phd](https://www.stcharleshealthcare.org/providers/marta-rowh-md-phd))
  - Emergency Medicine Physician at St Charles Health System
  - Other notes of interest: Research interest in public health and infectious disease
  - Also willing to discuss: Mentoring, Children, Atypical Trajectories
- **Daniel Wollman** ([https://www.danburyhospital.org/find-a-doctor/daniel-wollman](https://www.danburyhospital.org/find-a-doctor/daniel-wollman))
  - Geriatric Specialist at Danbury Hospital
  - Other notes of interest: Research interest in healthcare delivery, decision making
  - Also willing to discuss: Navigating the healthcare economy in less traditional ways
  - Assistant Professor, History and Philosophy of Medicine and Department of Internal Medicine, Division of Palliative Medicine
  - Other notes of interest: Research interest in ethics, history, disability
  - Also willing to discuss: Advocacy, Children, Negotiation, Job search in a time of crisis

Careers as Surgeon-Scientist

- **Peter Gruber** ([https://medicine.yale.edu/profile/peter_gruber/](https://medicine.yale.edu/profile/peter_gruber/))
  - Professor of Surgery; Vice Chair of Research for Surgery at Yale School of Medicine
- **Jason Wertheim** ([https://medicine.arizona.edu/person/jason-wertheim-md-phd](https://medicine.arizona.edu/person/jason-wertheim-md-phd))
  - Vice Dean, Scientific Initiatives; Associate Professor of Surgery and Biomedical Engineering at University of Arizona College of Medicine
  - Also willing to discuss: Mentoring
  - Team Leader at RIKEN Center for Integrative Medical Sciences in Japan
  - Also willing to discuss: Research outside US, General surgery residency


**Careers in Academia - Professorship**

- **Stuart Lipton** ([https://www.scripps.edu/faculty/lipton/](https://www.scripps.edu/faculty/lipton/))
  - Professor, Departments of Molecular Medicine and Neuroscience; Co-Director, Neuroscience Translational Center at Scripps Research Institute
  - Adjunct Professor, Departments of Neurosciences at UCSD School of Medicine; Department of Neurology at Yale School of Medicine
  - Other notes of interest: Developed FDA-approved drugs for Alzheimer’s disease
  - Also willing to discuss: Career path, Mentoring, Making discoveries, Managing lab and life

- **Marcela Maus** ([https://www.massgeneral.org/cancer-center/clinical-trials-and-research/center-for-cancer-research/investigators/maus-lab](https://www.massgeneral.org/cancer-center/clinical-trials-and-research/center-for-cancer-research/investigators/maus-lab))
  - Assistant Professor of Medicine at Harvard Medical School
  - Director, Cellular Immunotherapy Program at Mass General Hospital
  - Also willing to discuss: Mentoring, Children

- **Ralph DeBerardinis** ([https://profiles.utsouthwestern.edu/profile/99018/ralph-deberardinis.html](https://profiles.utsouthwestern.edu/profile/99018/ralph-deberardinis.html))
  - Professor, Children’s Medical Center Research Institute at UT Southwestern
  - Chief, Division of Pediatric Genetics and Metabolism
  - HHMI Investigator

- **Thao Nguyen** ([https://people.ctsi.ucla.edu/institution/personnel?personnel_id=7901177](https://people.ctsi.ucla.edu/institution/personnel?personnel_id=7901177))
  - Assistant Professor of Medicine at UCLA
  - Other notes of interest: Called as a witness for the prosecution in the pretrial and trial of Michael Jackson’s death
  - Also willing to discuss: Mentoring, Advocacy, Children, Women Physician-Scientists

- **Tamar Gur** ([https://u.osu.edu/gur.2/dr-gur/](https://u.osu.edu/gur.2/dr-gur/))
  - Assistant Professor, Psychiatry & Behavioral Health, Neuroscience, and Obstetrics and Gynecology at The Ohio State University College of Medicine
  - Assistant Director, MSTP, The Ohio State University College of Medicine
  - Also willing to discuss: Work-life integration