HARVARD
FAS Division of Science

science education

THE NINTH ANNUAL

Harvard Undergraduate Research Opportunities in Science (HUROS) Fair



ABSTRACTS

November 15, 2018
Harvard University

HARVARD
FAS Division of Science

science education

THE NINTH ANNUAL

Harvard Undergraduate Research Opportunities in Science (HUROS) Fair

Abstracts

November 15, 2018
Harvard University
Cambridge, MA

Table of Contents

Welcome to the HUROS 2018	3
Science Research Opportunities for Undergraduates: FAQ	5
Index	7
Abbreviations	9
Poster Abstracts	10
Informational Tables	100
Acknowledgements	109

Welcome to the 2018 Harvard Undergraduate Research Opportunities in Science (HUROS) Fair

What is HUROS?

The HUROS is a poster session where Harvard's science research groups explain their research to undergraduates and discuss possible undergraduate research projects.

What is Science Research?

Contemporary research in the sciences spans boundaries across traditional disciplines such as biology, chemistry, mathematics, statistics, physics, astrophysics, earth and planetary sciences, psychology, medical sciences, computer science, and engineering. For example, some researchers employ computational and chemical techniques to elucidate the structures of biological molecules; others strive to solve an engineering challenge with inspiration from a biological organism; and yet others investigate stem cells with a goal of developing therapies for human diseases.

What is the purpose of the HUROS?

This purpose of HUROS is to:

- ⇒ Provide a time and space for researchers from all of the Harvard's campuses and Harvard-affiliated research laboratories to speak in person with Harvard undergraduates interested in science research.

- ⇒ Connect research mentors with undergraduates interested in joining a lab or research group.

- ⇒ Promote Harvard programs, offices and organizations that administer fellowships, funding programs, and summer research internships that encourage undergraduate research and organize research conferences.

Which groups are here today?

The 2018 poster session includes researchers from the Faculty of Arts & Sciences, School of Engineering and Applied Sciences, Harvard Medical School, Harvard School of Dental Medicine, Harvard School of Public Health, Massachusetts General Hospital, McLean Hospital, Brigham and Women's Hospital, Boston children's Hospital, and more. We are grateful for the participation of these research groups and their support of undergraduate research.

Some of the other nearly 2,000 research labs at Harvard may have research opportunities for undergraduates, but are not able to attend the 2018 fair. See our website at http://lifesciences.fas.harvard.edu/harvard-affiliated-labs for more comprehensive listings of research areas and laboratories at Harvard. If you need help navigating your research path, you can set up an individual meeting with the Science Undergraduate Research Advisor, Dr. Anna Babakhanyan (ababakhanyan@fas.harvard.edu).

How do I navigate the HUROS?

Introduce yourself to the poster presenters and ask them to explain their research. Think about their research, hot it may relate to what you already know and ask questions.

Most of the posters are organized alphabetically by last name to encourage you to browse a number of different research areas. You may discover a new area or approach that captivates your interest. Finally, we have number of informational tables that provide information regarding summer research opportunities and funding.

Who sponsors the HUROS?

The Science Education Office in the Division of Science and the Office of Undergraduate Research and Fellowships (URAF) jointly sponsor this conference. We also thank the Harvard College Undergraduate Research Association (HCURA) for their participation.

Science Research Opportunities for Undergraduates: FAQs

lifesciences.fas.harvard.edu/research

Where can I do research?
There are nearly two thousand science research laboratories at Harvard. They are located not only at the Cambridge campus (Faculty of Arts & Sciences and School of Engineering and Applied Sciences), but also at Harvard Medical School, Harvard School of Public Health, and Harvard-affiliated hospitals and research institutions. See a list at http://lifesciences.fas.harvard.edu/harvard-affiliated-labs.

When can I start an independent research project in a laboratory or research group?
Most students dedicate the first semester of their first year getting acclimated to college life, college courses, and extracurricular activities. Some students join a lab the second semester of their first year or the summer after first year, while many others begin independent research during their sophomore year. Some students may start even later; however, if you intend to complete a senior thesis, plan to join a research group at the latest by the beginning of your junior year.

How can I find a research group to join?
A good place to start is to peruse the information in the Research tab of the Science Education website at http://lifesciences.fas.harvard.edu. Go to *Harvard-affiliated Labs* in the Research tab to find links to Harvard-affiliated research groups. For personalized advice, the Science Undergraduate Research advisor, Dr. Anna Babakhanyan (ababakhanyan@fas.harvard.edu) is here to help you define your research interests, find prospective labs, create a science-focused resume, write letters of inquiry to faculty, prepare for meetings with professors, and discuss funding or course credit options.

Do I need previous experience doing research?
You don't necessarily need previous experience to join many of Harvard's research labs. Most research groups are willing to train and mentor undergraduates who have limited previous lab or field research experience. Over time you will gain the skills and knowledge you need for an independent project. Also, most students acquire basic laboratory

skills in the laboratory sections of their science courses, and these help you transition into a research environment.

Can I earn course credit for term-time lab research?
Yes. The requirements for course credit vary among the different science and engineering concentrations, so it's best to contact a Concentration Advisor for specific details. The contact information for each of the Concentration Advisors is on the Life Sciences Education website in the Concentrations tab. For other concentrations, please contact the respective department.

Can I be paid for doing research during the summer or term-time?
Yes, but note that you can't simultaneously get paid and earn course credit for your research. For summer research, there are many sources of funding. Go to http://lifesciences.fas.harvard.edu and select *Research Opportunities* in the Research tab to see a list of funding opportunities. Notable funding sources include the Harvard College Research Program (HCRP) and the Faculty Aide Program. In addition, if you are eligible for the Federal Work-Study Program, you can qualify for term-time and summer research funding.

For more information or to make an appointment:
Please contact the Science Undergraduate Research Advisor, Dr. Anna Babakhanyan at ababakhanyan@fas.harvard.edu to schedule an advising meeting. She can help prepare for the interviews, provide information about funding and help with fellowship proposals and more.

Visit the Science Education Website at:
http://lifesciences.fas.harvard.edu/harvard-affiliated-labs

Index of Posters by Research Area

(numbers represent poster numbers and NOT page numbers)

Anesthesiology and Pain Medicine – 43, 62, 63, 88

Astrophysics – 95

Bioinformatics/Computational Biology – 1, 23, 24, 37, 41, 45, 50, 52, 56, 59, 60, 64, 65, 68, 69, 71, 73, 78, 83, 93, 97, 98, 99, 100, 102, 103, 104

Biomedical Engineering – 7, 10, 12, 13, 20, 24, 36, 56, 59, 61, 66, 67, 68, 70, 71, 74, 80, 81, 84, 85, 86, 88, 90, 91, 92, 93, 96, 97, 101, 102

Cancer – 5, 7, 12, 14, 15, 19, 57, 69, 75, 76, 81, 82, 85, 86, 90, 91, 98, 102, 106

Cardiovascular – 4, 36, 66, 73, 80

Chemistry – 3, 8, 12, 16, 44, 51, 53, 57, 58, 63, 65, 79, 88, 90, 9596, 98

Dental Medicine - 30

Diabetes – 30, 33, 46, 50, 70

Earth and Planetary Science – 3, 60, 69

Ecology – 3, 60

Endocrinology – 7, 40, 79, 82, 96

Engineering – 1, 3, 24, 36, 41, 45, 56, 58, 60, 66, 68, 69, 70, 74, 78, 80, 88, 92, 93, 95, 103

Genetics – 2, 15, 17, 19, 30, 34, 38, 44, 48, 50, 52, 53, 54, 70, 73, 75, 83, 86, 90, 91, 94, 96, 102, 106

Gerontology – 1, 30, 32, 61

Global Health – 13, 26, 27, 36, 51, 55

Immunology – 15m 18m 19m 46m 57m 62m 84m 85

Kidney Disease – 62

Mathematics – 1, 23, 24, 37, 41, 45, 50, 52, 56, 59, 60, 64, 65, 68, 69, 71, 73, 78, 83, 90, 93, 97, 98, 99, 100, 102, 103, 104

MCB – 4, 5, 7, 14, 17, 28, 29, 30, 35, 38, 39, 41, 47, 48, 53, 54, 62, 67, 73, 75, 77, 82, 83, 84, 86, 89, 92, 94, 96, 101, 102

Medicine – 2, 4, 6, 7, 10, 11, 12, 14, 15, 16, 17, 18, 19, 25, 29, 31, 32, 33, 34, 38, 40, 42, 46, 47, 48, 49, 50, 51, 55, 57, 61, 62, 63, 65, 67, 70, 72, 73, 75, 76, 77, 79, 80, 82, 87, 88, 90, 91, 96, 97, 98, 99, 106

Microbiology – 18, 19, 28, 51, 54, 101

Nanotechnology – 58

Neuroscience/ Neurology – 1, 2, 5, 6, 10, 20, 22, 27, 29, 31, 32, 37, 39, 44, 45, 48, 56, 64, 65, 66, 72, 75, 76, 77, 83, 87, 90, 94, 99, 100, 104

Nutrition – 9, 27, 70

OEB – 21, 34, 35, 42, 59, 61, 71, 89

Orthopedic – 25, 96

Ophthalmology – 11, 42, 55, 79, 97

Otolaryngology – 17, 20, 23, 31, 74, 88

Pediatric Medicine – 6, 13, 27, 36, 51, 80, 90, 104, 106

Pharmacology - 16, 51, 53, 57, 65, 79, 88, 90, 96, 98

Physics – 6, 10, 12, 20, 22, 24, 25, 31, 43, 45, 56, 60, 64, 65, 66, 67, 71, 74, 76, 78, 79, 80, 87, 88, 92, 93, 95, 99, 100, 104

Psychology/Psychiatry – 2, 16, 23, 27, 43, 45, 65, 72, 75, 83, 87

Public Health/ Epidemiology – 9, 13, 26, 67, 70, 98

Radiology/Imaging – 6, 10, 12, 22, 24, 25, 31, 43, 56, 64, 65, 66, 67, 71, 76, 78, 79, 80, 87, 95, 99, 100, 104

Statistics – 9, 50, 52, 56, 60, 88, 98

Stem Cell – 39, 40, 41, 49, 61, 70, 73, 75, 82, 85, 86, 89, 103, 106

Surgery – 6, 36, 49, 56, 63, 70, 88

Abbreviations

BCH: Boston Children's Hospital

BCMP: Dept. of Biological Chemistry & Molecular Pharmacology, HMS

BIDMC - Beth Israel Deaconess Medical Center, HMS

BME: Biomedical Engineering

BWH: Brigham and Women's Hospital

CCB: Chemistry and Chemical Biology

CPB: Chemical and Physical Biology

CSB: Computational and Systems Biology

HDRB: Human Developmental and Regenerative Biology

HEB: Human Evolutionary Biology

HMS: Harvard Medical School

HSPH: Harvard School of Public Health

HST: Health Sciences and Technology

IDI: Infectious Disease and Immunology

MCB: Molecular and Cellualr Biology

MEEI: Massachusetts Eye and Ear Infirmary

MGH: Massachusetts General Hospital

MGH/HST Athinoula A. Martinos Center for Biomedical Imaging, Massachusetts General Hospital, Harvard Medical School

OEB: Organismic and Evolutionary Biology

PSYCH: Psychology

NB: Neurobiology

SCRB: Stem Cell and Regenerative Biology

Poster Abstracts

(arranged by the faculty last name, see map for poster location)

Poster 1. Global Quantification of the Structural Brain Connectivity via a Conductance Model

Presenters: Aina Frau-Pascual, Iman Aganj
Principle Investigator: Iman Aganj, iman@nmr.mgh.harvard.edu
MGH/HST Athinoula A. Martinos Center for Biomedical Imaging, Massachusetts General Hospital, Harvard Medical School
Laboratory for Computational Neuroimaging Athinoula A. Martinos Center for Biomedical Imaging 149 Thirteenth Street, Suite 2301 Charlestown, Massachusetts 02129, https://www.martinos.org/lab/lcn
Contact: afraupascual@mgh.harvard.edu

Connectomics has proved promising in quantifying and understanding the effects of development, aging and an array of diseases on the brain. In this work, we propose a new structural connectivity measure that allows us to incorporate direct brain connections as well as indirect ones that would not be otherwise accounted for by standard techniques and that may be key for the better understanding of function from structure. From our experiments on the Human Connectome Project dataset, we find that our measure of structural connectivity better correlates with functional connectivity than streamline tractography does, meaning that it provides new structural information related to function. Through additional experiments on the ADNI-2 dataset, we demonstrate the ability of this new measure to better discriminate different stages of Alzheimer's disease. Our findings suggest that this measure is useful in the study of the normal brain function and structure, and for quantifying the effects of disease on brain structure. Number of hours per week: Negotiable Requirements: Proficiency in programming (MATLAB or python)

Poster 2. Increasing UBE3A in Glutamatergic Neurons of VMHvl Elevate Aggression Behavior

Presenters: Yi Nong, Morgane Boillot
Principle Investigator: Matthew Anderson mpanders@bidmc.harvard.edu
Beth Israel Deaconess Medical Center, HMS
CLS-630 330 Brookline Avenue, Boston, MA 02115, USA,
Contact: ynong@bidmc.harvard.edu

Individuals with autism spectrum disorder (ASD) are prone to behavioral difficulties including excessive tantrums, irritability, and self-injurious aggressive behaviors that often require medical treatment. Maternal

15q11-13 triplication triples the neuron-expressed dosage of UBE3A that encodes an E3 ubiquitin ligase and transcriptional co-regulator. Here we report that the transgenic mouse model of maternal 15q11-13 triplication (Ube3a2x mice), with increased copies of the Ube3a gene, displays increased aggression behaviors in addition to their impaired sociability and increased repetitive self-grooming. To map the neuronal circuits where increased UBE3A elevates aggression, we developed "LoxTBUbe3a" transgenic mice whereby UBE3A can be selectively increased in specific neuronal cell types and brain areas when combined with specific Cre driver mouse lines. Male mice with selective increases of UBE3A in glutamatergic neurons displayed elevated aggression (VGluT2Cre). In contrast, selective increases of UBE3A in GABAergic (VGATCre) did not affect indices of aggression. Prior work has shown that glutamatergic neurons in the ventromedial hypothalamus, ventrolateral subdivision (VMHvl) can drive attack behavior in male mice. To evaluate the role of VMH neurons in UBE3A-elevated aggression, LoxTBUbe3a-2x (homozygous for LoxTB-Ube3a) alleles were combined with the Sf1-Cre allele ,which expresses mainly in VMH). These male mice also displayed elevated aggression. We increased Ube3a selectively in glutamatergic neurons of the VMHvl by stereotactically injecting AAV-hSyn-DIO-Ube3a into VMHvl of VGluT2-Cre mice. These mice displayed heightened aggression compared with WT male mice injected with control AAV. These results indicate that increasing UBE3A in glutamatergic neurons of VMHvl is sufficient to elevate aggression. Number of hours per week: It is negotiable. Requirements: n/a

Poster 3. Design and Build Cutting-edge Instruments for Atmospheric Chemistry and Climate Applications

Presenters: Jessica Smith
Principle Investigator: James Anderson, anderson@huarp.harvard.edu
Research Associate, Harvard University School of Engineering and Applied Sciences
12 Oxford St., Link Bldg., Cambridge, MA 02138
https://www.arp.harvard.edu/
Contact: jsmith@huarp.harvard.edu

Research in the Anderson Group integrates science and engineering in service to society. Projects in this group offer unique opportunities for students to develop analytical and technical skills while designing and building cutting-edge instrumentation to solve real-world problems. The group specializes in the application of a wide range of spectroscopic techniques to measure key molecular species in the atmosphere aboard

high altitude aircraft and balloons. The measurements are then used to examine the relationships between the chemical composition of the Earth's atmosphere and the climate system. Specific examples of projects include 1) utilizing the Arduino microcontroller architecture, and 2) adapting innovative circuit design and control theory to build the next generation of instruments. Engagement with these projects will provide students exposure to problem definition, performance measurement, quantitative analysis, modeling, generation of creative solutions, engineering design trade-offs, and documentation/communication skills.
Number of hours per week: We would follow the above recommendations for number of hours per week
Requirements: No prior research experience is required, but preference will be given to students that have completed coursework in mechanical, electrical and/or optical engineering

Poster 4. cGMP-dependent protein kinase I in smooth muscle cells protects against stroke in male and female mice.

Presenters: Maria Shvedova, Dmitriy Atochin
Principle Investigator: Dmitriy Atochin, atochin@cvrc.mgh.harvard.edu
Massachusetts General Hospital, Cardiovascular Research Center, Division of Cardiology, Department of Medicine, Harvard Medical School, Charlestown, MA, United States of America
149 East, 13th Street, 4th floor, CVRC, Charlestown, MA, 02129, http://cvrc.massgeneral.org/faculty/dmitriy-atochin-phd/
Contact: atochin@yahoo.com

Cyclic guanosine monophosphate (cGMP)-dependent pathways have become increasingly important for screening of potential therapeutic targets in the context of brain ischemia/reperfusion injury. Although numerous studies devoted to cGMP-dependent protein kinase I (cGKI) have been conducted, the molecular mechanisms of its action in ischemia/reperfusion injury are not well understood. Our study presents novel data concerning cerebral ischemia/reperfusion injury in conditional cGKI knockout mice. Using an efficient tamoxifen-inducible smooth muscle cell-specific cGKI knockout mouse model, we showed that cGKI in smooth muscle cells renders a protective effect during ischemic stroke injury. Moreover, this protective effect of cGKI was independent of mouse gender and mediated at least in part via vasodilation. These results suggest that cGKI in smooth muscle cells should be targeted in studies designed to protect brain tissue against stroke.
Number of hours per week: It is negotiable, and depends on arrangement with each individual student.

Requirements: No prior research experience is required

Poster 5. Stearoyl CoA Desaturase is an essential regulator of fatty-acid-induced ER stress and a therapeutic target in glioblastoma

Presenters: Isam Adam
Principle Investigator: Christian Badr, badr.christian@mgh.harvard.edu
Isam Adam, Kelsey Pinkham, David Park, Arsalan Hashemiaghdam, Mohammad El-Abtah, Kamila Rosiak, Aleksander Kirov, Christian Badr
149 13th St, Charlestown, MA 02129
https://www.massgeneral.org/neurology/research/researchlab.aspx?id=1739
Contact: KPINKHAM@mgh.harvard.edu

Glioblastoma (GBM), the most common form of primary central nervous system tumors, has long been inherently difficult to treat and impossible to cure. These difficulties can be attributed to the presence of Glioma stem cells (GSCs) within the tumor and their innate ability to drive tumor progression, growth, and recurrence. Examining the metabolic activity, particularly that of fatty acids (FAs), in GSCs serves as a novel therapeutic approach to establishing new methods of treatment. Stearoyl CoA Desaturase I (SCD1) is a crucial enzyme in fatty acid metabolism through which conversion of saturated FAs into their unsaturated counterparts takes place. We have identified SCD1 as a critical enzyme that is essential for maintaining the survival and tumor initiating properties of GSCs which were found to be heavily dependent on this enzyme for tumor proliferation and survival. After knockdown of SCD1, it was found that tumor propagation was halted altogether, reinforcing GSC dependability on lipid synthesis. Pharmacological inhibition of SCD1 using a highly potent small-molecule inhibitor of SCD1 achieved a strong therapeutic outcome in two GSCs mouse models and was able to cure several mice bearing brain tumors. SCD1 is primarily localized in the endoplasmic reticulum (ER), and Fatty acids are known to impact ER stress thus hinting at a direct link between SCD1 activity and ER stress. Indeed, we report that the inhibition of SCD1 activity causes a toxic accumulation of Saturated fatty acids which cause an overwhelming ER stress response, promoting apoptotic cell death in GSCs. In summary, our study demonstrates an essential role for FAs in GSC maintenance and tumor initiation. We provide preclinical evidence for targeting lipid synthesis pathways in GSCs using potent inhibitors of SCD1 as a therapeutic treatment for GBM.
Number of hours per week: Negotiable: depends on arrangement with each student. Requirements: No prior research experience required.

Poster 6. Quantitative 3D MRI Evaluation of Infant Brain Size in the Context of Surgery and Critical Illness: The Impact of Gestational Age

Presenters: Chandler Mongerson, Dusica Bajic
Principle Investigator: Dusica Bajic, dusica.bajic@childrens.harvard.edu
Boston Children's Hospital
1 Autumn St. AU 338.1
http://www.childrenshospital.org/research/researchers/b/dusica-bajic
Contact: dusica.bajic@childrens.harvard.edu

Objective. To determine brain magnetic resonant imaging (MRI) measures of cerebrospinal fluid (CSF) and total brain size of full-term and premature infants following surgical treatment for noncardiac congenital anomalies requiring critical care. Methods. Full-term (n=13) and preterm (n=13) patients, and full-term naïve controls (n=19) < 1 year corrected age, underwent non-sedated brain MRI following completion of surgery and critical care treatment. Qualitative MRI findings were reviewed and reported by a pediatric neuroradiologist and neurologist. T2-weighted images were required for segmentation of CSF and total brain volumes using the Morphologically Adaptive Neonatal Tissue Segmentation (MANTiS) tool. Group differences in absolute (cm3) and normalized (as % total intracranial volume) data were analyzed with general linear model using age at scan as a covariate. Mean normalized values were assessed using one-way ANOVA. Results. Qualitative brain findings implicate brain atrophy in both full-term and preterm patients. Volumetric analysis confirmed significantly greater total CSF and extra-axial space, and decreased total brain size in both full-term and preterm patients compared to full-term naïve controls. In addition, volumetric analysis showed that normalized ventricular volumes were higher only in premature patients compared to controls. Conclusions. Full-term infants appear to have similar brain vulnerability in the context of surgical treatment of noncardiac congenital anomalies requiring critical care as premature infants. Future investigations of gray and white matter organization in at-risk full-term and premature infants should elucidate how perioperative critical care involving prolonged sedation can impact early brain development.
Number of hours per week: We recommend that during academic term (Sept-May) Freshmen and Sophomores work 6-10 hrs/week, Junior and Seniors 15-20 hrs/week. During Summer, all students are expected to work up to 40 hrs per week. Final hours are determined as per individual objectives and selected project.

Requirements: No prior research experience is required besides strong desire to learn something new and accomplish a research competency.

Poster 7. Leveraging reprogrammed metabolism to define selective vulnerabilities of IDH1 mutated cholangiocarcinoma cells

Presenters: Lei Shi
Principle Investigator: Nabeel Bardeesy
bardeesy.nabeel@mgh.harvard.edu
Center for Cancer Research, Mass General Hospital Department of Medicine, Harvard Medical School
185 Cambridge Street, CPZN 4100 Boston, MA 02114,
https://www.massgeneral.org/surgonc/research/researchlab.aspx?id=1161&display=overview
Contact: lshi7@mgh.harvard.edu

Cholangiocarcinoma is an epithelial malignancy of the biliary tract with poor prognosis that has been steadily rising incidence over the past several decades. Gain-of-function hot-spot mutations in the isocitrate dehydrogenase gene (IDH) are among the most common genetic alterations in these tumors. The mutant IDH1 enzyme produces high levels of the metabolite 2-hydroxyglutarate (2HG), which perturbs epigenetic control and cellular metabolism. We sought to further explore the metabolic processes altered by mutant IDH1 (and consequent abundance of 2HG) and to utilize this information to define selective vulnerabilities of IDH mutant cholangiocarcinoma cells that may inform novel therapeutic strategies. We show evidence that 2HG broadly reprograms cellular metabolism in cholangiocarcinoma cells, including prominent suppression of mitochondrial function and selective hinderance of the de novo pyrimidine synthesis pathway, which requires mitochondria. We integrated these findings with results from a series of screens that compared the sensitivity of IDH1 mutant and IDH1 wild type cholangiocarcinoma cells to extensive drug libraries. Among the most selective and potent hits in mutant IDH1 cells was an orphan compound that we found promoted an imbalanced nucleotide pool in IDH mutant cells, with overwhelming CTP levels compared to other nucleotides. In keeping with defective DNA replication and the generation of DNA damage associated with this imbalance, we observed activation of the ATM-TP53 DNA checkpoint pathway followed by apoptosis in these cells. Thus, we have uncovered alterations in nucleotide metabolism as central to the unique metabolic state of IDH1 mutated cholangiocarcinoma cells and demonstrated the potential of leveraging reprogrammed metabolism as a selective therapeutic strategy.

Number of hours per week: Negotiable, depends on individual student
Requirements: Requirements are to have interest, be focused while working and be responsible

Poster 8. Polynuclear Clusters: Electronic Structure, Multi-electron Reactivity and Magnetic Properties across the First Row
Presenters: Lisa Awaitey, Justin Teesdale
Principle Investigator: Theodore Betley, betley@chemistry.harvard.edu
Chemistry and Chemical Biology
Mallinckrodt 306 12 Oxford St Cambridge, MA 02138
http://betleylab.chemistry.harvard.edu/
Contact: awaiteyln@g.harvard.edu

In nature, polynuclear metalloenzyme cofactors (e.g., FeMoco in nitrogenase, oxygen evolving center in photosystem II) facilitate small molecule activation. The presence of multiple transition metal centers within close proximity to one another enables the complex to catalyze multi-electron redox processes, buttress redox changes within the cluster, and provide multiple coordination sites and modes for substrate binding. Understanding how these metal centers communicate with one another and perform these transformations is important for the design of synthetic mimics of these key enzymes. Previously, our lab has utilized a flexible polynucleating ligand to facilitate the isolation of tri- and hexanulcear transitional metal complexes. Taking advantage of the tunability of these synthetic systems, we are able to probe electronic structure-function relationships via X-ray crystallography, cyclic voltammetry, and NMR, IR, Mossbauer, and EPR spectroscopies. With a thorough understanding of the electronic structure of these clusters in hand, we aim to utilize their synthetic flexibility to perform reaction chemistry relevant to key biological transformations, providing further mechanistic understanding.
Requirements: No prior research experience is required

Poster 9. The VER-EAT!-TAS Initiative: Bringing Truth and Transparency to Everyday Food Choices for a Sustainable Future
Presenters: Stacy Blondin
Principle Investigator: Stacy Blondin, sblondin@hsph.harvard.edu
Harvard School of Public Health
677 Huntington Avenue, Boston, MA 02115, dining.harvard.edu/ver-eat-tas
Contact: sblondin@hsph.harvard.edu

Sustainable diets are patterns of eating that promote human health, food security, social justice, cultural diversity, and environmental and economic well-being for present and future generations. While we often receive messages about how our food choices affect our health and taste buds, we rarely receive information about the environmental, economic, and ethical implications of our choices. Yet, in order to nourish nearly 10 billion people by 2050, we need to collectively make more responsible food-related decisions. VerEatTas is an education initiative conceived of by researchers at the Harvard T.H. Chan School of Public Health, funded by the Harvard University Climate Change Solutions Fund, and developed in collaboration with the Office for Sustainability and Harvard University Dining Services. The goal of VerEatTas is to provide greater transparency about the food choices we make on campus every day, with the goal of positively impacting our health, the environment, and the way our food is produced. We welcome involvement from students who are interested in promoting dietary sustainability on Harvard's campus and/or college and universities nationwide through the Menus of Change University Research Collaborative. Specific roles could include: messaging and campaign development, data analysis, and/or thesis research. Please contact the project director and postdoc at HSPH, Dr. Stacy Blondin, with questions or statement of interest at sblondin@hsph.harvard.edu.
Number of hours per week: Term-time: minimum of 6 hrs/wk, maximum of 20 hours/week Summer: up to 40 hours/week
Requirements: While there are no specific qualifications, we would strongly prefer students with either/both a strong background in statistics and/or demonstrated interest in food system justice.

Poster 10. Microscopic Magnetic Stimulation of the Vagus Nerve: Initial Recordings

Presenters: Eric Milligan Ilknur Ay 2nd presenter, Sean Downs
Principle Investigator: Giorgio Bonmassar
Giorgio.Bonmassar@mgh.harvard.edu
(1) Athinoula A. Martinos Center, Massachusetts General Hospital, Charlestown, MA, United States (2) Harvard Medical School, Boston, MA, United States This work was supported by the US National Institutes of Health (NIH) grant R01MH111875.
75 13th Street, Charlestown, MA 02129, United States,
https://www.nmr.mgh.harvard.edu
Contact: eric_milligan@college.harvard.edu; IAY@mgh.harvard.edu

We have employed a novel type of stimulation, micro-magnetic stimulation (μMS), for vagus nerve stimulation (VNS). Electrical VNS is commonly used to treat drug-resistant epilepsy and depression. However, electrical VNS faces issues such as damaging oxidation/reduction reactions at the electrode-tissue interface, and concerns regarding device and tissue damage prevent patients with VNS implants from receiving the diagnostic benefits of MRI. μMS is accomplished by running current through a sub-millimeter coil to produce magnetic fields capable of inducing electric fields, which can lead to neuronal activation. μMS has high spatial resolution due to the small size of the coils involved. Unlike electrical stimulation, μMS can activate axons of a particular orientation based on coil orientation, and does not require charge-balanced stimulation waveform shapes to avoid electrophoresis. The coils used in μMS do not undergo oxidation/reduction reactions and are compatible with imaging devices such as MRI. Thus, μMS represents a groundbreaking step forward from electrical nerve stimulation. Healthy adult Wistar rats were used. The cervical vagus nerve was isolated and a microcoil was placed around the nerve. Arterial blood pressure (ABP), ECG, and respiration were recorded continuously before, during, and after cervical vagus nerve μMS. μMS of the vagus nerve caused a transient decrease in ABP and heart rate, indicating stimulation of the afferent and efferent vagal fibers. The findings are the first indication that μMS is capable of stimulating the vagus nerve. μMS also allowed the researchers much finer control over the strength and duration of the rats' physiological responses than electrical stimulation would have. This demonstrates the potential of μMS as a disruptive technology in the field of nerve stimulation.
Number of hours per week: Freshman and Sophomores: 6-10 Hours/Week Juniors and Seniors: 15-20 hours/week Summer hours: 40 hours/week Hours are negotiable, and depend on the arrangement with each individual student.
Requirements: Must be a full-time Harvard College Undergraduate Student.

Poster 11. Effects of vision impairment and age on driving
Presenters: Garrett Swan, Lily Zhang
Principle Investigator: Alex Bowers, alex_bowers@meei.harvard.edu
Schepens Eye Research Institute
20 Staniford St., Boston, MA 02114
http://bowerslab.eye.hms.harvard.edu
Contact: alex_bowers@meei.harvard.edu

Older drivers are the most rapidly increasing segment of the driving population. Continuing to drive in older age is very important to maintaining independence and a high quality of life. However, the onset of age-related vision or cognitive impairments may compromise driving safety. Currently the regulations regarding driving with impaired vision vary widely across the states and lack scientific basis. People with impaired vision can legally drive in some states but not others. Research at Bowers Lab focuses on quantifying the effects of different types of vision impairments on driving performance using a high-fidelity driving simulator. Driving performance is evaluated by measuring steering and lane-keeping skills and the detection of hazards in a timely manner. In addition to these measures, head and eye movements are recorded to better understand scanning (i.e. looking) behaviors while driving on highways and approaching intersections in city settings. We have ongoing studies evaluating the effects of cataracts, age-related macular degeneration, and hemianopia (the loss of half the field of vision following a stroke or traumatic brain injury) on driving performance. Our goals are to understand more about how each type of vision loss affects driving and how people with vision loss compensate for their impairment. In another set of studies we are evaluating the effects of different kinds of distraction on timely detection of hazards in the driving simulator and how the effects of distraction differ in younger and older drivers, with and without vision impairment.
Number of hours per week: Flexible
Requirements: No prior research experience

Poster 12. PET imaging of K+ channel expression of human tumor xenografts in mice: a potential novel biomarker in cancer
Presenters: Karla Ramos-Torres, Kazue Takahashi
Principle Investigator: Pedro Brugarolas, pbrugarolas@mgh.harvard.edu
Gordon Center for Medical Imaging, Department of Radiology
Massachusetts General Hospital/Harvard Medical School
55 Fruit St., Bulfinch 051 Boston, MA 02114
http://gordon.mgh.harvard.edu/brugarolas/
Contact: kmramos-torres@mgh.harvard.edu

Potassium channels are ubiquitous proteins involved in a wide variety of cell functions. While most commonly studied regarding propagation of action potentials, assessment of their roles in angiogenesis, cell proliferation and migration has propelled their emergence as potential biomarkers for cancer. Altered expression of voltage-dependent K+ (Kv) channels is found in several types of cancer cells and has been

correlated to tumorigenic state and degree of malignancy, suggesting the possibility of targeting potassium channels as tumoral biomarkers. Visualization of K+ channels via PET can be achieved with [18F]3-fluoro-4-aminopyridine ([18F]3F4AP), an analogue to K+ channel blocker 4-aminopyridine, recently developed to image changes in K+ channel expression in disorders like multiple sclerosis. Here we investigate this novel radioligand to image K+ channel expression in mouse xenograft models of glioblastoma, squamous lung cancer, and melanoma. [18F]3F4AP was synthesized according to published procedures and dynamic PET/CT imaging was performed after injection of the tracer. [18F]FDG was used for comparison. Results suggest differential tumor uptake of [18F]3F4AP when comparing the different cancer lines. Ratios of tumor-to-muscle signal show high uptake in glioblastoma, moderate in melanoma and low in lung cancer. In comparison, [18F]FDG imaging shows high uptake in melanoma, moderate in glioblastoma and low in lung cancer. Immunohistochemistry and immunoblotting studies of Kv channels are underway to further investigate the correlation between channel expression and [18F]3F4AP signal in tumors. In summary, [18F]3F4AP is a potential tracer for imaging K+ channel expression in cancer, which may provide valuable information about tumorigenic state or malignancy beyond FDG.

Poster 13. Survival After Use of a Condom UBT Package in Women with Uncontrolled Postpartum Hemorrhage in India

Presenters: Daniela Suarez-Rebling, Alicia Lightbourne
Principle Investigator: Thomas F. Burke, tfburke@mgh.harvard.edu
Massachusetts General Hospital, Harvard Medical School, Harvard T.H. Chan School of Public Health
125 Nashua Street, Suite 910 Boston, MA 02114
www.mghglobalhealth.org
Contact: dsuarez-rebling@mgh.harvard.edu

Introduction: Uncontrolled postpartum hemorrhage (PPH) is the leading cause of maternal mortality and morbidity. Timely intervention is critical. Over the past 8 years, we have developed and refined the award-winning package, Every Second Matters-Uterine Balloon Tamponade (ESM-UBT), designed to arrest uncontrolled PPH. The authors previously reported on high survival rates among women with uncontrolled PPH associated with use of an ESM-UBT device in Kenya, Tanzania, Sierra Leone and Nepal. This study examined the outcomes of women who had ESM-UBT devices placed for uncontrolled PPH across 10 medical colleges in Maharashtra, India. Methods: Data were collected

prospectively on all women with uncontrolled PPH who received ESM-UBT devices for any reason. Shock class was assigned based on recorded blood pressures and mental status at the time of UBT placement. Class III shock was defined as uncontrolled PPH and a systolic blood pressure (SBP) ≤ 90 mmHg and > 70 mmHg and/or altered mental status. Class IV shock was defined as uncontrolled PPH and SBP ≤ 70 mmHg and/or unconsciousness. Results: 149 women had an ESM-UBT device placed between January 2017 and February 2018, 119 (79.9%) of whom had uncontrolled PPH from uterine atony. 115 of the 119 (96.6%) women survived overall. 83 (69.8%) women had normal vital signs or were in class I/II shock, 28 (23.5%) were in class III shock, and 8 (6.7%) were in class IV shock. Survival rates were 100% (normal vitals or class I/II shock), 96.4% (class III shock), and 62.5% (class IV shock) respectively. Conclusion: Use of an ESM-UBT device for uncontrolled postpartum hemorrhage from atonic uterus is associated with high survival rates, especially when placed prior to class IV shock. Additional studies are needed to evaluate the feasibility and impact of ESM-UBT.Number of hours per week: Negotiable, depends on arrangement with student. Requirements: Interest in global health with a focus on maternal and child global health.

Poster 14. Investigating the mechanisms by which HSP70 supports mammary tumor metastasis

Presenters: Benjamin Lang, Patrick Magahis
Principle Investigator: Stuart Calderwood, scalderw@bidmc.harvard.edu
Dept. Radiation Oncology, Beth Israel Deaconess Medical Center
3 Blackfan circle, Center for Life Science, Room 617-F4,
https://connects.catalyst.harvard.edu/Profiles/display/Person/53611
Contact: bjlang@bidmc.harvard.edu

The stress inducible Heat Shock Protein 70 (HSP70) is clinically associated with breast cancer progression and supports several tumorigenic processes including oncogenic signaling, inhibition of apoptosis and aversion of senescence. HSP70 also modulates tumor metastasis, however the mechanisms that underlie this relationship remain to be established. To address this, our study aims to identify the major metastatic pathways that are dependent upon HSP70. RNA-seq and gene-ontology analysis of murine mammary tumors revealed a series of extracellular matrix (ECM)-encoding genes to be significantly lower in tumors derived from MMTV-PyMT Hsp70-/- mice. Our current studies focus on identifying the mechanisms by which HSP70 regulates the expression of ECM genes, and how HSP70-regulated ECM genes

influence tumor cell biology. Number of hours per week: Freshmen and Sophomores work 6-10 hours/week Juniors and Seniors 15-20 hours/week Summer students 40h/week Negotiable. Requirements: Interest in cell biology, molecular biology research, no prior research experience is required

Poster 15. Regulation of anti-lymphoma responses by a new HLA-E-related gene

Presenters: Lei Wang
Principle Investigator: Harvey Cantor, harvey_cantor@dfci.harvard.edu
Dana-Farber Cancer Institute
450 Brookline Avenue, Smith Research Labs, Room 722, Boston MA 02215, https://www.dana-farber.org/find-a-doctor/harvey-cantor/
Contact: alison_angel@dfci.harvard.edu

Insight into interactions between tumor cells and cellular components of the tumor microenvironment (TME) of malignant lymphoma has paved the way to novel and effective immunotherapies. The non-classical HLA-E molecules (Qa-1 in mice) represent an important immunologic checkpoint that may prevent successful attack by T cells and natural killer (NK) cells. However, the genetic and molecular basis for HLA-E-dependent regulation of immune responses to lymphoma has not been studied. To date, the major focus of the lymphoma response has been effector T cells, while studies of CD8 regulatory T cells (CD8 Treg) and NK cells have been scanty. To explore the contribution of Qa-1 to the generation and activation of CD8 Treg and NK cells, we have generated EL4 lymphoma cell lines with mutations in Qa-1 that delete the two functional loci – T23 and T11. The T11 gene represents a novel recently-discovered component of the Qa-1 locus. Although both T11 and T23 encode functional MHC class Ib molecules that share peptide-presenting activity, only T23 engages the inhibitory NKG2A receptor on NK cells. We use these and other Qa-1 mutant lymphoma lines to interrogate the separate regulatory contributions of Qa-1-T23 and Qa-1-T11 to the anti-tumor immune response and the effects of Qa-1 blockade. We are testing the hypothesis that T11 expression by tumor, unlike its paralog T23, enhances the host anti-tumor response and loss of T11 class Ib MHC expression may confer a selective advantage to tumors. This study will establish a foundation to investigate T23 as well as newly discovered T11-dependent control of the immune response.
Number of hours per week: During the academic year, a minimum of 15-20h and set schedule is recommended, exclusive of exam periods. Requirements: Experience is preferred, but not essential.

Poster 16. Effects of morphine abstinence on oxycodone self-administration in male and female rats

Presenters: Tania Lintz, Suman Guha
Principle Investigator: Elena Chartoff, echartoff@mclean.harvard.edu
McLean Hospital, Harvard Medical School
McLean Hospital 115 Mill Street MRC 218, https://www.chartofflab.com/
Contact: tlintz@mclean.harvard.edu

Opioid abuse often begins after withdrawal from prescription opioid painkillers. Opioid withdrawal includes intense, short-lived physical symptoms and protracted psychological symptoms, which contribute to addictive behavior. Women are often more likely than men to abuse opioids to self-treat negative affect and pain, suggesting that withdrawal-induced negative affective states could increase abuse risk more in women. Opioid abuse is associated with heightened levels of distress intolerance - defined as the perceived inability to tolerate negative physical (e.g. pain) and emotional states - raising the possibility that distress intolerance can be used to predict the likelihood of initiating prescription opioid abuse. Here we used acoustic startle, warm water tail flick latency, and somatic withdrawal signs as proxies for distress intolerance in adult male and female rats after a regimen of escalating dose morphine injections. After 2 weeks of abstinence, rats were implanted with jugular vein catheters and allowed to self-administer oxycodone. We found that morphine withdrawal was associated with decreased tail flick latencies in male, but not female, rats—indicative of hyperalgesia. In addition, somatic withdrawal signs were increased in both sexes, whereas no change in acoustic startle was observed. Male, but not female, morphine-withdrawn rats initially self-administered more oxycodone than vehicle-withdrawn rats, suggesting that morphine withdrawal increases vulnerability to initiate opioid abuse specifically in males. However, after extended oxyodone exposure, intake normalized across groups. Individual distress intolerance measures were correlated with acquisition of oxycodone self-administration, supporting our hypothesis that this approach may be useful in predicting vulnerability to initiating opioid abuse.
Number of hours per week: Negotiable and depends on the individual student and the project(s) to be done.
Requirements: Willingness and some evidence of competency in handling rats and rat behavior.

Poster 17. Restoration of Hearing by CRISPR/Cas9 mediated genome editing in the Pmca2 deafness mouse model by protein delivery

Presenters: Veronica Lamas, Wan Du
Principle Investigator: Zheng-Yi Chen
Zheng-Yi_Chen@meei.harvard.edu
Department of Otology and Laryngology, Harvard Medical School, Massachusetts Eye and Ear Infirmary
243 Charles St, Boston, MA, 02114, USA
https://scholar.harvard.edu/chenlab
Contact: Zheng-Yi_Chen@meei.harvard.edu

One in 500 newborns suffers from genetic hearing loss, and hundreds of genes are likely to be responsible for genetic hearing loss when mutated. CRISPR/Cas9-mediated genome editing presents an opportunity to develop new type of treatment for genetic hearing loss by permanent editing that targets the mutations to restore hearing. However, most CRISPR/Cas9 has been performed in germline or in vitro by viral vectors or DNA vectors, which raise long-term safety concerns. Further in vivo efficiency has been generally low. To develop CRISPR based gene therapy to treat genetic deafness we explored direct in vivo protein:RNA complex delivery into a genetic deafness mouse model, Oblivion (Obl), due to a mutation in the Pmca2 gene, and assess the effect on hearing restoration. Direct protein:RNA complex injection in vivo led to dramatic hearing recovery across most frequencies. Hearing recovery was the most significant in the middle frequencies, by as much as 45dB. Hearing recovery was gRNA specific and dose-dependent. Application of Cas9:gRNA resulted in rescue of inner ear hair cells, which are lost to the Pmca2 mutation. Auditory neurites were persevered after Cas9:gRNA delivery, whereas in the control Obl mice a majority of them underwent degeneration.

Poster 18. Clostridium difficile toxins induce VEGF-A and vascular permeability to promote disease pathogenesis

Presenters: Qianyun Lin
Principle Investigator: Xinhua Chen, xchen1@bidmc.harvard.edu
Harvard Medical School Beth Israel Deaconess Medical Center
330 Brookline Ave, DA-609 Beth Israel Deaconess Medical Center,
https://connects.catalyst.harvard.edu/Profiles/display/Person/57042
Contact: xchen1@bidmc.harvard.edu

Clostridium difficile infection (CDI) is mediated by two major exotoxins, toxin A (TcdA) and toxin B (TcdB), which damage the colonic epithelial barrier and induce inflammatory responses. The function of the colonic vascular barrier during CDI has not been studied. Here we report increased colonic vascular permeability in CDI mice and elevated vascular endothelial growth factor A (VEGF-A) which was induced by infection with a TcdA and/or TcdB-producing strain in vivo but not with a TcdA-TcdB- isogenic mutant. TcdA or TcdB also induced VEGF-A in human colonic mucosal biopsies. Hypoxia-inducible factor (HIF) signaling appeared to mediate toxin-induced VEGF production in colonocytes, which can further stimulate human intestinal microvascular endothelial cells. Neutralization of VEGF-A and inhibition of its signaling pathway each attenuated CDI in vivo. Compared to healthy controls, CDI patients had significantly higher serum VEGF-A, which subsequently decreased after treatment. Our findings indicate critical roles for toxin-induced VEGF-A and colonic vascular permeability in CDI pathogenesis. It may also implicate the pathophysiological significance of gut vascular barrier in response to virulence factors of enteric pathogens. As an alternative to pathogen-targeted therapy, this study may enable new host-directed therapeutic approach for severe, refractory CDI.
Number of hours per week: Negotiable depending on arrangement with individual student

Poster 19. Genomics and proteomics of host-adenovirus interactions

Presenters: Ashrafali Mohamed Ismail, Jaya Rajaiya
Principle Investigator: James Chodosh, Jaya_rajaiya@meei.harvard.edu
Department of Ophthalmology, Howe Laboratory, Massachusetts Eye and Ear, Harvard Medical School, Boston, MA.
Massachusetts Eye and Ear Infirmary 243 Charles street Boston MA 02114, https://www.hms.harvard.edu/dms/virology/fac/chodosh.php
Contact: mohamed_ismail@meei.harvard.edu

Human adenovirus (HAdV) has played an important role in the study of human biology, including the discoveries of RNA splicing and viral oncogenesis. The Encyclopedia of DNA Elements (ENCODE) project illustrated functional elements in the majority of the human genome. We hypothesized novel, previously unidentified, gene coding functions for HAdVs. Using in silico TRANSFAC analysis, we identified potential site-specific DNA binding transcription factors (TFs) for HAdV-D37, a major ocular pathogen. OCT4, a key regulator in embryonic development and stem cell pluripotency, was identified to significantly bind HAdV-D37

DNA. To map the HAdV-D37 genome-wide occupancy of OCT4, chromatin immunoprecipitation and high throughput sequencing (ChIP-Seq) were performed in infected A549 cells. In subsequent knockdown and overexpression experiments, OCT4 was shown to variably transactivate or repress specific HAdV-D37 early genes. Further studies of TF binding may reveal new mechanisms in viral gene transcriptional regulatory processes for better understanding of virus-host co-evolution. Concurrent with our interest, we also show potential host proteins that interact with minor coat protein pIIIa for the first time. We developed an inducible expression system for pIIIa to reproduce expression levels at near endogenous levels after infection. By affinity purification and LC-MS/MS analysis we identified two high confidence interactors of pIIIa: ubiquitin-specific protease 9x (Usp9x) and nucleoporin Nup358. Together, these host proteins may assist in nuclear export and virus assembly. Further investigation of molecular interactions during viral infection may uncover novel roles for viral encoded proteins in the host cellular environment. Number of hours per week: work hours is negotiable. Requirements: No prior research experience is required

Poster 20. Understanding—and Fixing—the Inner Ear

Presenters: David Corey
Principle Investigator: David Corey, dcorey@hms.harvard.edu
Harvard Medical School
Goldenson 443, 220 Longwood Ave, Boston, MA 02115, USA,
http://corey.med.harvard.edu/
Contact: dcorey@hms.harvard.edu

We work on a fundamental problem in sensory neuroscience: how the vibration of sound is converted to an electrical signal by receptor cells of the cochlea. These cells, called hair cells for the tuft of mechanosensitive cilia on their top surface, open a set of ion channels when the cilia are moved by sound, and the resulting ionic current depolarizes the cells to initiate neurotransmitter release. We know many of the proteins in the mechanotransduction complex, but don't know how they connect to each other or how they change shape in response to mechanical force. Our work involves a combination of single-cell biophysics, cryo-electron microscopy and protein biochemistry; it has the ultimate goal of understanding the complex both structurally and biophysically, at an atomic level. All of these proteins are required for sensation of sound, and so it is not surprising that mutations in many of them cause hereditary deafness. We use mouse models of hereditary deafness, in which one or another gene for these proteins is

nonfunctional, and we use new viral vectors to deliver replacement genes to the hair cells. In several of these models, we can partially rescue the deafness in mice. The ultimate goal will be to develop gene therapy methods for treating hereditary deafness in humans. Number of hours per week: time negotiable. Requirements: laboratory experience preferred, but mostly intelligence and dedication

Poster 21. Testing the bet-hedging hypothesis: is intragenotypic variability in thermal preference adaptive?

Presenters: Jamilla Akhund-Zade, Denise Yoon
Principle Investigator: Benjamin de Bivort, debivort@oeb.harvard.edu
OEB
Northwest Labs 248, debivort.org
Contact: jakhundzade@g.harvard.edu

Individuals differ in their behavior – there exists a wide range of behavioral differences between individuals that are consistent across environments and time in every species examined. We have found that these behaviors are manifested in isogenized lines reared identically - we label these differences intragenotypic variability. Bet-hedging is an evolutionary strategy that is optimal in environments with high environmental variance. Under bet-hedging, a single genotype encodes for multiple phenotypes, which can be a plausible explanation for the existence of variability. We are testing the hypothesis that thermal preference variability is different across geographic regions in North America due to the differing advantages of bet-hedging strategy. We are gathering wild fly strains from different locations from across the U.S.A. with the help of collaborating labs in order to test this hypothesis. In addition, we are examining how mean thermal preference changes over the course of the season by collecting and assaying local wild flies. To elucidate the proximal causes of thermal preference variability, we are looking at whether individual differences in the expression of a thermosensor correlate with thermal preference. Identifying the molecular factors underlying differences in phenotypic variability will provide us with new capacity to understand the mechanisms of phenotypic evolution. Number of hours per week: 5-10 hours/week (negotiable)
Requirements: Some prior experience with molecular biology techniques (e.g. DNA extraction or PCR) is preferable, but not required. We will consider all interested applicants!

Poster 22. Inverse Association Between Lateral Ventricle Volume And Fractional Anisotropy Of The Corpus Callosum In First Episode Schizophrenia

Presenters: Sarah Haferman
Principle Investigator: Elisabetta del Re
elisabetta_delre@hms.harvard.edu
Harvard Medical School
Contact: elisabettacdelre@gmail.com

Background. The volume of the central corpus callosum (CCC) is inversely correlated with that of bilateral lateral ventricles (LV) in first episode schizophrenia (FESZ) (del Re, 2016). Here we determined corpus callosum (CC) diffusion measures (DTI) using two tensor tractography in FESZ and controls in order to investigate the relationship between diffusion in CCC and LV volume at baseline and 0.7 months later. Freewater correction was applied to account for the possible contribution of LV CSF to CC DTI measures. Methods. Sixteen FESZ and 16 age and handedness matched controls were included in the study. MR images were acquired on a 3Tesla whole body MRI Echo speed system General Electric scanner (GE Medical Systems, Milwaukee) at Brigham and Women's Hospital at baseline and ~0.7 years later with 51 gradient directions (1.7 mm slice thickness). The CC was segmented in five regions according to Hofer and Frahm, 2006. Clinical and demographic data were collected. Results. Fractional anisotropy (FA) was decreased, radial diffusivity and trace were increased while axial diffusivity was unchanged in FESZ with comparison to controls. In FESZ an inverse correlation was present between FA of the CCC and volume of the LV; in controls the correlation was also significant but positive. In FESZ an inverse correlation was present between lower FA of the CCC and higher positive symptoms. Conclusions. In FESZ, the FA of CCC and the volume of LVs are inversely correlated and lower CCC FA associates with higher positive symptoms. Schizophrenia specific developmental mechanisms might determine this relationship. Number of hours per week: 10 hrs Requirements: no prior research experience is required.

Poster 23. Neural coding of complex sounds in normal hearing and in hearing with cochlear implants

Presenters: Yoojin Chung
Principle Investigator: Bertrand Delgutte
bertrand_delgutte@meei.harvard.edu
Massachusetts Eye and Ear, Harvard Medical School

243 Charles St. Boston MA,
http://research.meei.harvard.edu/neuralcoding/
Contact: yoojin_chung@meei.harvard.edu

The long-term goal of our group's research is to determine how speech and other biologically-significant sounds are processed by the auditory system, with a focus on understanding the neural mechanisms underlying auditory perception. To achieve this goal, we combine three major techniques of integrative neuroscience: neurophysiological (single-unit recordings), computational (mathematical models) and psychological (through collaborations with psychoacousticians). Ongoing research is focused in two major directions: (1) studies of the neural mechanisms of spatial hearing and pitch perception in the auditory brainstem, and (2) studies aimed at improving perceptual performance with cochlear implants, particularly binaural implants. These lines of research are related in that, while normal-hearing listeners excel at understanding speech in noisy and reverberant environments, hearing-impaired and cochlear-implant patients often have difficulties in these adverse environments, even if they do well when facing a single speaker in a quiet room. To understand these differences between normal listeners on the one hand, and hearing impaired listeners and cochlear implant users on the other hand will require an integrated knowledge of the auditory processing of speech, binaural processing in the auditory brainstem, and how these mechanisms are altered by development and pathology. Such knowledge will be invaluable for a rational design of improved hearing aids, auditory implants and rehabilitation strategies.Requirements: basic computer skills, necessary software training will be given. No prior research experience is required.

Poster 24. Artificial Intelligence Empowering Patients and Clinicians
Presenters: Sehyo Yune, Hyunkwang Lee
Principle Investigator: Synho Do, sdo@mgh.harvard.edu
25 New Chardon St. Ste 400, Boston MA 02114
http://lmic.mgh.harvard.edu/

At the Laboratory of Medical Imaging and Computation of Massachusetts General Hospital, we are working at the intersection of computer science, big data, clinical medicine, and population health. As a multidisciplinary team of data scientists, machine-learning engineers, biomedical engineers and physicians, we develop tools using artificial intelligence (AI) technologies that bridge the gap in the clinical practice

and the healthcare delivery system. Our research involves development of machine-learning algorithms that i) detect critical conditions from medical images, ii) expedite clinical workflow, iii) improve reliability of clinical tests, and iv) quantifying clinically relevant measures to be used in clinical decision making. We have developed a bone age assessment model that achieved an accuracy over 98%, a muscle segmentation model which showed a dice similarity coefficient of 0.93, a catheter recognition system that locates a catheter tip within 3 mm error, and a brain hemorrhage detection model of which the sensitivity was 98% and the specificity was 95%. In addition to medical image analysis, our research also includes development of natural language processing (NLP) models to automatically label radiology data, extract relevant features from electronic health records (EHR), and build clinical decision support systems. We are expanding our scope to build prediction models and blockchain-based health information exchange platforms.

Poster 25. Research opportunity in the Q-MIG lab

Presenters: Alexander Mathiessen
Principle Investigator: Jeffrey Duryea, jduryea@bwh.harvard.edu
BWH, Thorn Building, 333C-D 75 Francis St, Boston, MA 02115

Objective: To examine the impact of structural changes on loss of cartilage and knee function in patients with knee osteoarthritis (OA). Background: OA is the most common joint disease, affecting millions of adult Americans. There is no proven treatment to reverse joint damage from OA. Tracking the trajectory of structural changes well in advance of meaningful outcomes is critical to establish modifiable targets for prevention or treatment. Plain radiographs are insensitive and nonspecific to critical changes in joint structure. •MRI detects structural changes that may be early signals of future OA and potential targets for treatment - but the expense of acquiring and reading MRI data has limited its potential to smaller studies. Project description: • In this multi-center study, we have access to the world's largest (n=4796) longitudinal (96mo) knee OA cohort with available MRI-images (Osteoarthritis Initiative). The project will use highly efficient computational methods to automatically and quantitatively measure OA-related knee structures on MRI. The results of this study will provide new insights into the prognosis, prevention, and treatment of knee OA, with the ultimate goal of facilitating the development and assessment of highly needed disease-modifying drugs for OA. Learning outcomes: Study design, data assessment, and analysis methods, scientific writing. Opportunities for co-authorship depending on performance Funding:

Students are encouraged to apply to the HCRP and other fellowships or register for a research course credit. Limited funding may be available from the lab. Application: Students should submit a resume and what their goals are in pursuing this research.

Poster 26. Eclampsia in Africa: who, where and when?
Presenters: Eziamaka Ezenkwele, Ifeanyi Menuba
Principle Investigator: Eziamaka Ezenkwele
eezenkwele@hsph.harvard.edu
T. Chan Harvard School of Public Health University of Nigeria, Nsukka University of Nigeria Teaching Hospital, Enugu

Objectives: to measure the incidence of eclampsia, determine the type of health facility eclamptics were referred from, the gestational age at first seizure and whether there is seasonal variation or not. Methodology: A retrospective study of 42 eclamptics at University of Nigeria Teaching Hospital, Enugu, Nigeria from 2004 to 2011 was done. Information collected from the case notes included sociodemographic characteristics, booking status, type of health facility referred from, gestational age at first seizure, month of admission and maternal deaths. Results: The hospital incidence of eclampsia was 0.86%. The mean age was 25.8 years ±9.6 years. Fifty percent were between 20 and 29 years, more than half primigravidae (57.1%), and 31% had secondary school education: these observations were statistically significant (x2 = 17.05, P <0.01, x2 = 27.14, P <0.01, x2 =11.33, P = 0.01 respectively). Majority (85.7%) were unbooked; this was statistically significant (x2=21.43, P = 0.00). Thirty three percent were referred from a private hospital and 26.2 % from a maternity home. The mean gestational age at first seizure was 35 weeks ± 4 weeks. Eighty-three percent of the seizures occurred during the rainy season; this was statistically significant (x2 =18.67, P <0.01). The case fatality rate was 14.3%. Conclusion: Eclampsia is one of the leading causes of maternal mortality. Majority of the eclamptics were primigravidae, unbooked and sought care at substandard facilities. There is need for health education of women of child bearing age as well as their families.

Poster 27. The relationship between biological and psychosocial risk factors and resting-state functional connectivity in 2-month-old Bangladeshi infants
Presenters: Theodore Turesky
Principle Investigator: Nadine Gaab nadine.gaab@childrens.harvard.edu
Boston Children's Hospital Harvard Medical School

1 Autumn St. Boston, MA 02215, thegaablab.com
Contact: theodore.turesky@childrens.harvard.edu

Childhood poverty has been associated with structural and functional alterations in the developing brain. However, poverty does not alter brain development directly, but is thought to act through biological or psychosocial risk factors associated with extremely low income-to-need ratios (e.g., malnutrition, infection, low maternal education). Yet few studies have investigated risk factors in the context of early infant brain development, and none have done so in low resource settings, where children are often exposed to multiple risk factors. Resting-state fMRI data were acquired in 16 infants from families living in extreme poverty and 17 infants from more affluent families in Dhaka, Bangladesh. Whole-brain intrinsic functional connectivity (iFC) was performed using bilateral seeds in amygdala, where iFC has shown susceptible to early life stress, as well as sensorimotor areas, which have exhibited less susceptibility to early life stress. Infants from very poor families compared to those from more affluent families exhibited greater (i.e., less negative) iFC in the precuneus for seeds placed in amygdala; however, no significant group differences were observed in iFC for seeds placed in sensorimotor areas. Height-for-age (a proxy for malnutrition and infection) was not associated with amygdala/precuneus iFC; in contrast, maternal education was correlated with amygdala/precuneus iFC and family conflict during pregnancy mediated this relationship, suggesting that psychosocial risk factors commonly associated with poverty are also related to brain function in the amygdala. The present study expands upon prior studies of poverty by examining infants exposed to extreme biological and psychosocial risk factors.
Number of hours per week: During the academic year, students are required to commit to at least 10 hours/week and during the summer they must commit to 8 weeks full-time (this is negotiable).
Requirements: No prior research experience is required. Coursework in Neuroscience/Psychology and experience working with infants/children will be helpful.

Poster 28. Characterizing the molecular mechanisms for how strain identity regulates social swarming behavior in the bacterium Proteus mirabilis

Presenters: Achala Chittor
Principle Investigator: Karine Gibbs, kagibbs@mcb.harvard.edu
Department of Molecular and Cellular Biology, Faculty of Arts & Sciences, Harvard University

Biological Laboratories, 3rd floor
https://www.mcb.harvard.edu/directory/karine-gibbs/
Contact: chittor@g.harvard.edu

Surfaces can be hospitable homes to many bacterial communities. For the opportunistic pathogen *Proteus mirabilis*, rigid surfaces are a platform on which this organism is induced to elongate up to 40-fold in length and rapidly spread out, with populations of cells ("swarms") covering centimeters within a few hours. Swarm migration can reportedly occur on hard nutrient surfaces, e.g., up to 2.5% agar and latex. Moreover, kin recognition behaviors are apparent in P. mirabilis swarms; genetically distinct isolates remain spatially separated on surfaces. Swarm motility is associated with virulence, and kin recognition factors are implicated in mixed-species infections. We have previously identified and characterized several key proteins involved in recognition behaviors. However, how these proteins interact and how recognition contributes to population separation and swarm motility are largely undetermined. Moreover, the intersections between kin recognition, local nutrient availability, and cellular metabolism remain unclear, especially in terms of regulating a cell's decision to migrate in a swarm or to stay put in an attached colony. We propose that strain-specific information, exchanged among neighboring cells, regulates how *P. mirabilis* forms and maintains surface-dwelling communities such as swarms. *P. mirabilis* causes infections of the bladder and is thought to be able to invade via migration along a catheter through the urinary tract. We aim to build better models for how *P. mirabilis* colonizes the bladder, and by extension how to prevent colonization, by examining the contributions of kin recognition, surface conditions, and local environmental nutrients.
Number of hours per week: Hours are negotiable and depends on arrangement with each individual student.
Requirements: No prior research experience is required.

Poster 29. Activating proteasomes to remove toxic proteins and combat neurodegenerative diseases
Presenters: Jordan VerPlank, Galen Collins
Principle Investigator: Alfred Goldberg
alfred_goldberg@hms.harvard.edu
Harvard Medical School, Department of Cell Biology
240 Longwood Ave C-411 Boston MA, 02115,
https://agoldberg.med.harvard.edu/
Contact: jordan_verplank@hms.harvard.edu

The vast majority of intracellular proteins in eukaryotes are degraded by the 60-subunit molecular machine called the proteasome. The proteasome specifically degrades proteins tagged with chains of ubiquitin. It is commonly believed that the rate of ubiquitination determines proteins' rates of degradation. However, we and other labs recently uncovered mechanisms of regulating proteasome function that alter the rate of intracellular protein degradation. Our lab is interested in how the proteasome and protein degradation is controlled in both normal and disease conditions. There is growing evidence in neurodegenerative diseases like Alzheimer's disease and Amyotrophic Lateral Sclerosis (ALS) that degradation by the proteasome is impaired and misfolded, toxic proteins accumulate and contribute to the pathogenesis and disease progression. Thus, enhancing the ability of the proteasome to destroy these toxic proteins has the therapeutic potential to combat these diseases. Our lab has uncovered several mechanisms that enhance proteasome function, including the binding of adaptor proteins to proteasomes and the phosphorylation of proteasome subunits. Excitingly, proteasome phosphorylation can be promoted by various pharmacological agents, and we've found that FDA-approved drugs that activate kinases improve mouse and zebrafish models of several neurodegenerative diseases. Number of hours per week: Negotiable Requirements: No prior research required.

Poster 30. Molecular, cellular and genetic basis of skeletal homeostasis and its regulation in health and disease

Presenters: Francesca Gori
Principle Investigator: Francesca Gori
francesca_gori@hsdm.harvard.edu
Harvard School of Dental Medicine, Division of Bone and Mineral Research
188 Longwood Ave, Boston, MA
Contact: francesca_gori@hsdm.harvard.edu

The longstanding interest of our laboratory is to understand the molecular, cellular and genetic basis of skeletal homeostasis and its regulation in health and disease. Our current projects can be subdivided into the following areas: 1) Wnt signaling and bone. WNT signaling is one of the most important developmental signaling pathways that control cell fate decisions and tissue homeostasis. Ongoing studies on the role of WNT signaling antagonists and agonists in skeletal homeostasis are part of the current focus of the lab. 2) Hippo signaling in skeletal homeostasis and responses to mechanical loading. The Hippo/YAP-TAZ

signaling, involved in various mechanical cues with implications for cell fate, tissue development and homeostasis, has been recently implicated in bone biology. Ongoing studies focus on the role of this signaling in skeletal homeostasis. 3) Osteocytes and skeletal homeostasis. Osteocytes, the most abundant cells in bone, are embedded into bone and in intimate contact with the bone matrix. Ongoing studies explore the link between the matrix that surrounds the osteocytes and the way in which they regulate bone remodeling and/or mineral metabolism. 4) Mechanisms by which PTH/PTHrP regulates skeletal homeostasis. Ongoing studies focus on the exploration of novel molecular pathways by which PTH regulates MSC fate decision and favors bone formation while repressing marrow adipogenesis. 5) Central regulation of energy homeostasis and bone formation. Ongoing studies are focused to identify the precise neuronal circuits and the cellular and molecular pathways, regulating energy homeostasis and bone formation. These studies address an important clinical challenge for aging related metabolic disorders (obesity, diabetes mellitus type 2 and osteoporosis).
Number of hours per week: 10 hs/wk. Requirements: no prior research experience is required

Poster 31. Tracking the neural dynamics and decoding the neural representations that support and structure spoken language
Presenters: Seppo Ahlfors, Adriana Schoenhaut
Principle Investigator: David Gow, gow@helix.mgh.harvard.edu
Harvard Medical School. Massachusetts General Hospital (Department of Neurology)
Neurodynamics and Neural Decoding Group (Gow), rm. 2030, 100 Cambridge Street, Boston, MA, 02114, (currently under construction)
Contact: seppo@nmr.mgh.harvard.edu

The Neurodynamics and Neural Decoding Group (Gow Lab) develops and applies sophisticated analytic tools to understand the mechanisms responsible for the lawful sound structure of spoken words and syllables (phonology). Our research examines the role of interactive pattern association mechanisms versus generative grammatical processes in shaping phonology and phonotactic processes. Our methods begin with the creation of high spatiotemporal resolution reconstructions of event-related brain activity based on the integration of magnetoencephalographic (MEG), electroencephalographic (EEG) and magnetic resonance imaging (MRI) data. We use machine learning techniques to determine what kinds of information are encoded in localized patterns of brain activation (neural decoding), and a technique

called Granger Causation analysis to track patterns of directed information flow between these localized brain regions over large networks with millisecond temporal resolution. These methods address fundamental inferential limitations in cognitive neuroscience, linguistics, and cognitive psychology. We are looking for student researchers who are interested in advancing theory by helping design, implement and analyze new experiments aimed at testing bedrock premises of generative linguistics, classical computational accounts of cognition, and emergentist approaches inspired by connectionism and dynamic systems theory. While it is not a requirement, we would be extremely interested in working with students with fluency in Hebrew or Arabic to help us explore critical linguistic puzzles that are specific to the Semitic languages. We would also be very interested in working with students with strong coding skills (MatLab/Python) who might be interested in helping us develop, refine, and test our cutting-edge processing stream. Number of hours per week: Negotiable. Minimum of 8 hours per week (academic year and summer), with maximum of 40 hours per week (summer only). Requirements: No prior experience necessary, but background in experimental psychology or linguistics, and basic coding skills are highly desireable. Fluency in Hebrew or Arabic would be extremely valuable, but is not required.

Poster 32. Alpha-synuclein and Tau abnormalities caused by lipids in neurodegenerative aging

Presenters: Oeystein Brekk
Principle Investigator: Penny Hallett, phallett@mclean.harvard.edu
McLean Hospital/Harvard Medical School
115 Mill St. Belmont, MA 02478,
https://www.mcleanhospital.org/biography/penny-hallett
Contact: obrekk@mclean.harvard.edu

Aging is the most significant risk factor for developing genetic and sporadic neurodegenerative disorders, including Parkinson's disease (PD) and Alzheimer's disease (AD). The connections between aging and disease-associated gene products, for example GBA1, alpha-synuclein (aSYN), or Tau, are not fully understood. We have previously observed age-dependent reductions in glucocerebrosidase (GCase) activity (encoded by GBA1) in normal aging in the human (Rocha et al., 2015, Ann. Clin. Trans. Neurol.) and mouse (Hallett & Huebecker et al., 2018, Neurobiol. Aging) brain, with concurrent accumulation of a variety of glycosphingolipids (GSLs) including glucosylceramide and glucosylsphingosine. As glucosylceramide has recently been shown to

induce aSYN aggregation and attenuate neurotoxicity in vitro (Zunke et al., 2018, Neuron), we wish to investigate the impact of age-related lipid impairments on disease-associated proteins aSYN and Tau. In wildtype (WT) mice aged 2-24 months of age, presenting with elevated GSL content, we investigated lipid modifications of aSYN and Tau through biochemical lipid-extraction assays of whole-brain homogenates and assessed how these modifications affect protein structure and posttranslational state. Using fluorescent labeling and automated colocalization analysis, we determined the localization of these proteins within the neuronal lipid membrane compartment with known lipid membrane markers including vesicular membranes, to identify likely binding partners. Lastly, we will assess any correlation between GCase function and lipid homeostasis on aSYN burden in human sporadic PD. As an alternative to the classic view of proteinopathy, these findings illuminate the interplay between age-associated changes in lipid homeostasis and the risk for neurodegenerative disease.
Number of hours per week: As recommended and to be negotiated with the student. Requirements: No prior research experience is required

Poster 33. Relationship Between Frequency of Blood Glucose Testing and Reduction in HbA1c and Body Weight During Intensive Lifestyle Intervention in Patients with Type 2 Diabetes and Obesity
Presenters: Shaheen Tomah, Noor Mahmoud
Principle Investigator: Osama Hamdy, osama.hamdy@joslin.harvard.edu
Joslin Diabetes Center, Harvard Medical School
1 Joslin Pl, Rm 316 Boston, MA 02215, N/A
Contact: shaheen.tomah@joslin.harvard.edu

Self-monitoring of blood glucose (SMBG) is known to be valuable for proper diabetes management. However, the optimal frequency of SMBG has been debated. The aim of this study is to evaluate the relationship between the frequency of SMBG and the degree of glycemic control and the magnitude of weight reduction in patients with type 2 diabetes (T2D) and obesity enrolled in an intensive lifestyle intervention (ILI). We evaluated 36 patients with T2D and obesity (mean age 56±9 yrs, 56% female) enrolled in the Weight Achievement and Intensive Treatment (Why WAIT) program, a 12-week multidisciplinary ILI clinical program between May 2016 and December 2017. Participants were asked to test their blood glucose before each meal, at bedtime, before and after exercise, occasionally postprandial and when needed. At baseline, average body weight was 105.7±20.9 kg, BMI 36±6 kg/m2, HbA1c 7.8±1.3%, and 41% of participants were treated with insulin. Based on

their actual frequency of SMBG per day, participants were divided into tertiles. The lowest tertile tested on average 2.3±0.6 times/day (range: 1.1-2.9 times). The middle tertile tested on average 3.3±0.3 times/day (range: 3.0-3.9 times). The highest tertile tested on average 5.2±1 times/day (range: 4.0-7.7 times).HbA1c and body weight did not differ between tertiles at baseline. At 12 weeks, HbA1c changed by -0.9±0.3% (p=0.01), -1.2±0.4% (p<0.01) and -1.7±0.5% (p=0.001) respectively from baseline. Body weight changed by -7.6±3.2 kg (-7.13%), -9.2±6.4 kg (-8.7%), and -10.6±4.1 kg (-9.9%) respectively from baseline (p<0.01 for all). The reduction in HbA1c and body weight was significantly better in the highest tertile of SMBG in comparison to the lowest tertile (p<0.05 for both). In conclusion, patients with T2D and obesity who test their blood glucose more often during ILI achie
Number of hours per week: Negotiable
Requirements: No prior research experience is required

Poster 34. Genetic studies of body proportion (sitting height ratio) shed light on the recent evolution of human skeletal growth
Presenters: Eric Bartell, Rebecca Fine
Principle Investigator: Joel Hirschhorn
Joel.Hirschhorn@childrens.harvard.edu
Boston Children's Hospital
3 Blackfan Circle, Boston MA 02445,
http://www.childrenshospital.org/research/labs/hirschhorn-laboratory
Contact: ebartell@g.harvard.edu

Genome-wide association studies (GWAS) have identified hundreds of common genetic variants associated with human height; however, less is known about the genetics of body proportion. A greater understanding of the genetics of sitting height ratio (SHR, sitting height divided by standing height) would expand our understanding of skeletal growth. We use data from the UK Biobank to expand our genome-wide association study of SHR from ~25,000 to >400,000 individuals. Height-increasing alleles that decrease SHR were deemed to act primarily on the legs, and height-increasing alleles that increase SHR were deemed to act primarily on the head/spine. For analyses of selection, we used methods from our earlier work, and expanded study populations to include those genotyped in the China Kadoorie Biobank (CKB), constituting ~100,000 further individuals. We identified >1800 genome-wide significant SHR-, Height-, or both-associated variants (p<5x10-8). We used the SHR association data to classify height variants according to their upper or lower body effect. In preliminary analyses, the signal of selection

appears stronger in height-associated variants that act on long bones than in variants that act on spine/head length; if confirmed in the larger sample, this would indicate that recent evolutionary pressures differentially altered limb length within Europe. Results also show more differentiation in allele frequencies at loci that affect torso length in CKB populations as compared to leg length; this may argue for different selection pressures acting on the two different study populations. In conclusion, we have identified loci associated with SHR that offer new insights into the genetic, biological and evolutionary basis of body proportion and skeletal growth.
Number of hours per week: Negotiable
Requirements: Some biology or computer science or math background

Poster 35. Evolution of morphology and behavior in Peromyscus mice

Presenters: Nick Jourjine, Brock Woolridge
Principle Investigator: Hopi Hoekstra, hoekstra.hopi@gmail.com
MCB, Hoekstra Lab, Biological Labs, 16 Divinity Avenue,
https://hoekstra.oeb.harvard.edu/
Contact: nicholas_jourjine@fas.harvard.edu

What is the genetic basis of animal morphology and behavior? What are the mechanisms that drive morphological and behavioral evolution in natural populations? In the Hoekstra lab, we address these fundamental questions in a behaviorally diverse, but experimentally tractable, rodent model of behavioral evolution: the deer mice (genus Peromyscus). Peromyscus mice are among the most abundant mammals in North America, where they have diversified to occupy nearly every available ecological niche and have evolved a range of social systems. The natural history of Peromyscus has been documented extensively, and many species are inter-fertile, making it possible to link genetic variation to adaptive behavior in natural settings. They are also super cute. These features make Peromyscus an ideal system to ask questions at the intersection of evolutionary biology, animal behavior, and neuroscience, and the Hoekstra lab uses a variety of computational and experimental tools to answer these questions. Undergraduates interested in computer science, population genetics, and animal behavior are encouraged to apply for positions in our lab!
Number of hours per week: Freshmen and Sophomores: 6-10 hours/week during school semester, 40 hours/week during summer
Juniors and Seniors: 15-20 hours/week during school semester, 40 hours/week during summer

Requirements: No prior research experience is required.

Poster 36. Engineered lung device that supports premature infants for post-natal lung development

Presenters: David Hoganson, Pedro del Nido
Principle Investigator: David Hoganson
david.hoganson@cardio.chboston.org
Department of Cardiac Surgery, Boston Children's Hospital
Boston Children's Hospital 61 Binney St, Enders Research Building Lab #1322 Boston, MA 02115
http://www.childrenshospital.org/directory/physicians/h/david-hoganson
Contact: kimberlynrich15@gmail.com

A microfluidic engineered lung device has been developed that utilizes an engineered vascular network and adjacent alveolar chamber. Oxygen and carbon dioxide are exchanged across the 9 μm silicone respiratory membrane and the device achieves gas exchange equivalent to a modern cardiopulmonary bypass oxygenator. In an effort to develop a lung assist device to support premature infants achieve lung maturation development after birth, the silicone microfluidic vascular network is now being developed out of collagen thin film technology. Collagen thin films on the order of microns thick are pattern in the desired vascular network shape and used to create complex vascular networks capable of being endothelialized prior to clinical use. Current work is focused on the optimization of the vascular network mechanics, developing biomimetic combinations of extracellular matrix components for optimal endothelialization and tailoring device seeding and culture approaches. Interfacing the vascular network with small diameter vascular graft and evaluating in a small animal model are also being pursued to work towards this foundational advance in the development of engineered organs. Number of hours per week: 8 - 20 hours per week - negotiable full time summer position available. Requirements: Prior research experience is a plus. Engineering background, CAD/Solidworks experience, tissue culture experience and animal testing experience all would be beneficial but not required. There is also an opportunity to assist with the development of medical devices for cardiac surgery applications.

Poster 37. Abnormal amygdala activity during fear conditioning in subclinical psychosis

Presenters: Wisteria Deng
Principle Investigator: Daphne Holt, dholt@mgh.harvard.edu

Department of Psychiatry, Massachusetts General Hospital, Boston, MA; Athinoula A. Martinos Biomedical Imaging Center, Charlestown, MA
149 13th Street, Charlestown, MA 02129 http://holtlab.wixsite.com/esnlab
Contact: ywdeng@mgh.harvard.edu

Aberrant fear learning mechanisms have been identified in a variety of psychological disorders. These abnormalities may predict risk of psychosis or psychosis-like symptoms at a subclinical level. Here, we tested the hypothesis that increased levels of persecutory delusional thinking is related to alterations in the mechanisms of fear conditioning, with particular interest in the function of the amygdala. This was investigated in young adults with subclinical levels of psychosis-like symptoms, a population naïve to antipsychotics. We found that healthy youth without persecutory beliefs showed differential responses in anterior insula, superior frontal cortex, amygdala, hippocampus and default network areas (middle temporal cortex, precuneus and angular gyrus). In contrast, youth who endorsed persecutory beliefs showed abnormal responses in many of these areas during fear conditioning (i.e., they failed to show differential, conditioned fear responses in these regions). We further examined the subnuclei of the amygdala and found that the abnormalities in amygdala responses during fear conditioning in youth with persecutory beliefs were limited to three subnuclei: the central, accessory and corticoamygdaloid nuclei. Our findings suggest that there is an altered fear learning mechanism in the amygdala and closely connected regions in youth with psychotic-like experiences, particularly persecutory thinking.
Number of hours per week: 10-20 hours/week. Negotiable and flexible.
Requirements: Good interpersonal skills, maturity and problem-solving abilities. proficiency in Word, Excel and Powerpoint. Knowledge of MATLAB or UNIX (or similar programming languages) would be a plus (not required).

Poster 38. Highly Multiplexed FISH For In Situ Genomics

Presenters: Maristela Onozato, Clarence Yapp
Principle Investigator: John Iafrate, aiafrate@partners.org
Department of Pathology, Massachusetts General Hospital, Boston, MA
149 13th st., suite 6027, Charlestown, MA 02129,
Contact: monozato@mgh.harvard.edu

Background/Objective: The quantification of changes in gene copy number (CN) is critical to our understanding of tumor biology and for the clinical management of cancer patients. DNA fluorescence in situ

hybridization (FISH) is the gold standard method to detect CN alterations, but is limited by the number of genes one can quantify simultaneously. To increase the throughput of FISH technique, we devised a fluorescent ”barcode” system for the unique labeling of dozens of genes and an automated image analysis algorithm that enabled their simultaneous quantification. Methods: Locus-specific DNA sequences were bar-coded and assembled into a single probe mix. FISH spots were recorded with a multispectral fluorescence imaging system and quantified by a custom built imaging software. Traditional ‘gold standard’-single-gene-FISH (control) and aCGH were also performed to obtain the basic validation of the multiplexed FISH assay. Cell lines and CTCs successful analysis was defined by finding the expected copy number of each gene in each tested cell line. Results: Multiplex-FISH with automated copy number calls were accurate across all lines tested, with high-level amplification of the MYC gene detected as expected in lung cancer line H1975. Hybridizations with a higher plex level (>15 genes) demonstrated that the biggest hurdle is to minimize false negatives calls that can lead to some of the genes being under-represented during quantification. Conclusion: We developed an assay for automated quantification of gene copy number in pathology specimens. Our studies showed that accurate detection of dim FISH spots in the presence of non-noise disruptive features, and non-uniform background spectra are the major challenges that need to be addressed especially when dealing with formalin fixed paraffin embedded samples.
Number of hours per week: Hours will be negotiable and arranged with each individual student
Requirements: No prior research experience is required but previous exposure to a laboratory environment is preferable for safety reasons. The research involves a wet lab and a computer based components and the student can choose to work on both or either one.

Poster 39. Familial LRRK2 G2019S Parkinson’s disease patient fibroblasts have reduced mitochondrial clearance

Presenters: Ria Thomas
Principle Investigator: Ole Isacson, isacson@hms.harvard.edu
McLean Hospital/Harvard Medical School
McLean Hospital, 115 Mill St., Belmont, MA 02478,
www.neuroregeneration.org
Contact: rthomas@mclean.harvard.edu

Human induced pluripotent stem cell -derived neurons from LRRK2 G2019S mutation carriers exhibit disrupted mitochondrial movement and

increased vulnerability to chemical stressors of mitochondrial function (Cooper, Seo et al. 2012). We and others have also uncovered changes in mitochondrial phenotypes in fibroblasts derived from LRRK2 G2019S carrying PD patients. Utilizing a static co-localization based assay we have shown that LRRK2 inhibition normalizes LRRK2 mutation-mediated changes in mitophagy in patient fibroblasts (Smith, Jansson et al. 2015), but the dynamics of this process largely remain unknown. To study the temporal changes in mitophagy, we performed live cell imaging of mitophagic flux in human patient fibroblasts using a pH sensitive dual fluorescence reporter. Decreased rate of mitophagy was observed at baseline and upon treatment with a mitochondrial complex I inhibitor in PD patient fibroblasts carrying the LRRK2 G2019S mutation compared to healthy subject controls. Pharmacological and genetic inhibition of the LRRK2 kinase activity normalized this phenotype. Further investigation of the regulators of mitophagy pathway affected by this mutation showed that the cells carrying the LRRK2 G2019S mutation displayed a significant increase in the mitochondrial marker Tom20, mitophagy initiator Optineurin, lysosomal content regulators TFEB and LAMP1, and a decrease in autophagosome load associated LC3 I/II ratio. These data show that the LRRK2 G2019S mutation alters mitophagy, which may have important implications for understanding the underlying biology leading to pathology in vulnerable cells carrying the mutation.
Number of hours per week: To be negotiated with each student.
Requirements: No prior experience required

Poster 40. Endocrinology Undergraduate Research
Presenters: Rona Carroll
Principle Investigator: Ursula Kaiser
Brigham and Women's Hospital, Department of Medicine Endocrinology
http://researchfaculty.brighamandwomens.org/BRIProfile.aspx?id=1102,
Contact: rcarroll@bwh.harvard.edu

Our laboratory provides a rich intellectual environment of physicians and scientists focused on neuroendocrinology and reproductive endocrinology. An elaborate neural network integrating internal and external signals governs the onset of puberty and subsequent fertility. The precise nature and components of this network are not well established, but it is clear that puberty is triggered by the central increase in pulsatile gonadotropin-releasing hormone (GnRH) secretion, which stimulates the secretion of the pituitary gonadotropins, necessary for the activation of gonadal function. Our laboratory uses a translational approach to study the hormones and genes involved in the

neuroendocrine regulation of reproductive development, puberty onset and subsequent fertility. Out approaches include studies of: 1. Hypothalamic and pituitary cell models and in vitro studies; 2. In vivo mouse models; and 3. Patients with clinical reproductive disorders. Potential Student Roles: (1) Assist with reproductive phenotyping of genetically modified mouse models (e.g., pubertal markers, estrous cyclicity); (2) Assist with human genetic studies, including review of human patient clinical data to correlate with genetic findings with clinical phenotypes, and DNA sequencing and sequence analysis, including analyses of exome sequence data; (3) Contribute to laboratory studies of human inducible pluripotent stem cell (hiPSC)-derived hypothalamic neurons that have been genetically modified by CRISPR-Cas9.
Number of hours per week: negotiable
Requirements: Group is not attending HUROS, contact them by email if interested

Poster 41. Computational Tools for high-dimensional single cell analysis

Presenters: Sam Wolock, Caleb Weinreb
Principle Investigator: Allon Klein, allon_klein@hms.harvard.edu
Harvard Medical School Department of Systems Biology
200 Longwood Avenue - Warren Alpert 536, Boston, MA 02115,
https://klein.hms.harvard.edu/
Contact: Caleb_Weinreb@hms.harvard.edu

Every cell in your body has an almost identical genome, and yet these cells perform wildly different functions and come in many shapes and sizes. How can a single genome give rise to such diverse cell states? New tools for measuring gene expression in single cells may bring us closer to answering this question. But this type of data is entirely new in biology, and so the algorithms and computational techniques required to interpret it are still in their infancy. The goal of this summer project is to help develop computational tools that can make sense of high-dimensional single-cell measurements. The project will offer an opportunity to gain hands-on experience in cutting-edge computational biology including "big data" approaches, and to demonstrate the utility of the methods for studying stem cells. We are looking for a student with prior experience in software development, including knowledge of python and javascript, to help develop a visualization method called SPRING for interacting with high-dimensional single-cell gene expression data.

Number of hours per week: Commitment to the project can be flexible, though a minimum of 10 hours per week would be preferred. Some of the work can be done offsite from the HMS campus
Requirements: We are looking for a student with prior experience in software development, including knowledge of python and javascript.

Poster 42. The Evolution and Development of Complex Visual Systems

Presenters: Kyle McCulloch
Principle Investigator: Kristen Koenig, kmkoenig@fas.harvard.edu
OEB, Northwest Labs, RM 368 52 Oxford Street Harvard MA,
https://www.koenig-lab.com/

In the Koenig Lab, we are interested in how eyes evolved. We study this question from a cellular and developmental perspective. There is an incredible diversity of visual organs found across the animal kingdom, ranging from simple and imperfect eye spots to the most complex and elegant visual systems. Although we can observe this variation in form and function across animals, the molecular mechanisms underlying these differences remains largely unknown. We study the developmental basis of phenotypic diversity found across animal visual systems and are interested in how developmental processes evolve to generate this diversity. We use emerging sequencing techniques, Crispr/Cas9 genome editing, and advanced microscopy and live-imaging methods to understand how complex morphological structures are formed, how multiple cell types are generated, and the gene regulatory changes that underlie these processes. We use a comparative approach, studying the visual systems of many organisms across the tree with the hope of gaining insight into how complex organs evolve. Our current primary research models in the lab are the cephalopod, Doryteuthis pealeii and the starlet sea anemone, Nematostella vectensis.
Number of hours per week: Negotiable
Requirements: No prior research experience required.

Poster 43. Analgesic Effects Evoked by Real and Imagined Acupuncture: A Neuroimaging Study

Presenters: Jin Cao, Georgia Wilson
Principle Investigator: Jian Kong, jkong2@mgh.harvard.edu
Department of Psychiatry, Massachusetts General Hospital, Harvard Medical School
Building 120, 2nd AVE, Charlestown, MA, 02129,
https://connects.catalyst.harvard.edu/Profiles/display/Person/79124

Contact: GJWILSON@mgh.harvard.edu

Acupuncture can provide therapeutic analgesic benefits but is limited by its cost and scheduling difficulties. Guided imagery is a commonly used method for treating many disorders, such as chronic pain. The present study examined a novel intervention for pain relief that integrates acupuncture with imagery called video-guided acupuncture imagery treatment (VGAIT). A total of 27 healthy subjects were recruited for a crossover-design study that included 5 sessions administered in a randomized order (i.e., baseline and 4 different interventions). We investigated changes in pain threshold and fMRI signals modulated by: 1) VGAIT, watching a video of acupuncture previously administered on the participant's own body at baseline while imagining it being concurrently applied; 2) a VGAIT control condition, watching a video of a cotton swab touching the skin; 3) real acupuncture; and 4) sham acupuncture. Results demonstrated that real acupuncture and VGAIT significantly increased pain threshold compared with respective control groups. Imaging showed that real acupuncture produced greater activation of the insula compared with VGAIT. VGAIT produced greater deactivation at the rostral anterior cingulate cortex. Our findings demonstrate that VGAIT holds potential clinical value for pain management.
Number of hours per week: During the academic term: 6-10 hrs/week for freshmen and sophomores, 15-20 hrs/week for juniors and seniors. During the summer: 10-40hrs/week. Hours negotiable, and depends on arrangement with each individual student.
Requirements: no prior research experience is required.

Poster 44. Genetic Manipulations in the Fruit Fly Fight Club: Amine function examined at single neuron levels; differences between female and male aggression; genetic studies in hyper-aggressive "Bully" flies
Presenters: Saheli Sengupta, Rache Monyak
Principle Investigator: Edward Kravitz, edward_kravitz@hms.harvard.edu
Harvard Medical School
Department of Neurobiology, 220 Longwood Avenue, Boston, MA 02115, http://www.hms.harvard.edu/bss/neuro/kravitz/
Contact: Saheli_Sengupta@hms.harvard.edu

Aggression is an essential innate behavior that is used across the animal kingdom to acquire food, territory and mates. We developed a fruit fly model system to study aggression using powerful genetic tools that allow

us to: (i) reproducibly identify single neurons important in the behavior; and (ii) manipulate these neurons while animals are behaving normally. Male and female flies fight in same sex pairings with the males fighting at higher intensity levels and establishing hierarchical relationships and females fighting at lower levels and sharing resources. Animals learn during bouts and the outcomes of fights influence future behavior, with "winner" and "loser" effects seen. Neurohormones, including amines, peptides and classical transmitters, influence the behavior and are involved in circuitry associated with the behavior. By inbreeding winners of fights for 35 generations we generate "bullies" that fight at higher intensity levels and always win fights against the parent strain. Specific gene expression differences associated with this phenotype have been identified in the bullies. Serotonin facilitates going to higher levels of intensity in male fights, but is not required for aggression. Using an intersectional genetics approach, we found 2 pairs of serotonergic neurons responsible for the transition to high levels of aggression in males and identified downstream single neuron targets of these neurons. These influence aggression in opposite directions. Studies have been initiated examining circuit differences and similarities between male and female aggression. In these studies we identified a mutant strain in which females fight at intensity levels comparable to high intensity levels seen in males. At present we plan optogenetic studies of brain neuron function in freely moving flies. (supported by NIGMS)
Number of hours per week: Negotiable, juniors, seniors usually take Bio Research as a course, full time summers
Requirements: Intro Neurobiology; Intro genetics

Poster 45. Biological and Artificial Intelligence
Principle Investigator: Gabriel Kreiman
gabriel.kreiman@tch.harvard.edu
Children's Hospital, Harvard Medical School
1 Blackfan Circle, Karp 11217, http://klab.tch.harvard.edu
Contact: gabriel.kreiman@tch.harvard.edu

The Kreiman lab is interested in the neuronal circuits and algorithms responsible for visual object recognition and memory formation. Visual object recognition is crucial for most everyday tasks including face identification, reading and navigation. Episodic memories constitute the essential fabric of our recollections. The Kreiman lab combines neurophysiology, psychophysics and theoretical/computational modeling to understand the neuronal circuits, algorithms and computations

performed by the visual system and to develop biophysically-inspired computational approaches to machine vision and memory formation. Number of hours per week: 6-15 hours/week during the academic year 40 hours/week during the summer.Requirements: Computer programming experience is desired but not required

Poster 46. The Kuchroo Immunology Lab
Presenters: Alexandra Schnell, Rocky Barilla
Principle Investigator: Vijay Kuchroo,
vkuchroo@evergrande.hms.harvard.edu
Harvard Medical School and Brigham and Womens Hospital
60 Fenwood Road, Boston, http://kuchroolab.bwh.harvard.edu/
Contact: alexandraschnell@g.harvard.edu

We are an immunology lab that studies autoimmunity, which refers to an inappropriate immune system attack on the body's own tissues. It underlies many devastating human diseases, such as multiple sclerosis (MS), type I diabetes, rheumatoid arthritis and colitis. In the Kuchroo lab, we study the contribution of CD4+ T cells to the development of autoimmunity. In particular, we study experimental autoimmune encephalomyelitis (EAE), a mouse model of MS. Using transgenic and knockout mice, we aim to shed light on the genes and molecular pathways that control CD4+ T cell responses in EAE. For example, the lab has identified new cell surface receptors that can lead to the death or exhaustion of autoreactive T cells. Further, we have characterized novel CD4+ T cells called the Th17 cells which induce autoimmune tissue injury.

Poster 47. α-TAT1 mediates Microtubule Acetylation and Promotes Lung Fibrosis
Presenters: Alba Santos, Tobias Kuehl
Principle Investigator: David Lagares, DLAGARES@mgh.harvard.edu
Massachusetts General Hospital - Harvard Medical School
Charlestown Navy Yard (CNY) Building 149, Rm. 8.127 13th St.,
Charlestown, MA 02129
Contact: asantoshipolito@mgh.harvard.edu

BACKGROUND: Idiopathic pulmonary fibrosis (IPF) is a severe lung disease characterized by the deposition, remodeling, and stiffening of extracellular matrix buildup. This matrix stiffening promotes lung fibrosis by inducing mechanoactivation of fibroblasts via mechanotransduction pathways. Here we investigate how mechanical signaling modulates

microtubule acetylation to promote myofibroblast activation and lung fibrosis. METHODS: Mechanoactivation of human lung fibroblasts (HLFs) was induced by culturing these cells on soft (1kPa) or stiff (100kPa) polyacrylamide hydrogels. Mass spectrometry was used to identify possible post-translational modifications in the microtubule cytoskeleton. To overexpress αTAT1 - the enzyme that promotes microtubule acetylation - we engineering two retroviral vectors expressing wild type (WT) αTAT1 and a catalytically dead αTAT1 [D157N]. siRNA against αTAT1 was used for loss-of-function studies. Global αTAT1 KO mice were subjected to the bleomycin-induced lung fibrosis model. Masson´s trichrome staining, hydroxyproline content, and αSMA immunohistochemical staining were used to quantify the resultant lung fibrosis. Lung vascular permeability was assessed with Evan's blue assay. Percentages of immune cells (macrophages, neutrophils, and CD4+ and CD8+ T cells) were determined by cytofluorimetry. RESULTS: Mass spectrometry analysis revealed that HLFs cultured on stiff matrices (100kPa) showed hyper-acetylated α-tubulin at residue lysine 40 compared to cells cultured on soft matrices (1kPa). α-tubulin K40 acetylation is regulated by the enzyme αTAT1, which showed to induce myofibroblast activation. In vivo, global αTAT1 knock out mice were significantly protected from bleomycin-induced lung fibrosis. Number of hours per week: negotiable, depending on each individual student. Requirements: No prior research experience required

Poster 48. Proteostasis and Mitochondrial Pathways in Causes and Treatments of Parkinson's Disease

Presenters: Whitney Gibbs, Hailey Novis
Principle Investigator: Matthew LaVoie, mlavoie@rics.bwh.harvard.edu
Brigham and Women's Hospital and Harvard Medical School
HBTM Room 10012 60 Fenwood Road Boston, MA 02115,
http://lavoielab.bwh.harvard.edu/
Contact: mlavoie@rics.bwh.harvard.edu

The overall goal of the LaVoie lab is to elucidate the underlying pathophysiology of Parkinson's disease (PD). We consider not only the specific inherited gene mutations in familial forms of PD but also maintain a focus on the more common sporadic form of the disorder. Our study of familial PD is centered on pathogenic mutations in the Parkin and LRRK2 genes and our examination of idiopathic PD focuses on models of mitochondrial dysfunction. We employ a wide variety of tools in our work including genetic mouse models of PD, human neurons differentiated from patient-based stems cells, CRISPR/Cas9 gene

editing, and a diverse array of biochemical and microscopic techniques to dissect the molecular mechanisms of this neurodegenerative disease. Or hope is to translate discoveries surround the etiology of PD into opportunities to slow or halt the disease process in patients.
Number of hours per week: ~10hr/week during the year, full time over the summer is available. Requirements: no prior research experience necessary, cell culture/standard biochemistry a plus.

Poster 49. Using the mouse digit tip to understand human regeneration

Presenters: Gemma Johnson
Principle Investigator: Jessica Lehoczky, jlehoczky@bwh.harvard.edu
Brigham and Women's Hospital
Hale BTM 5022, 60 Fenwood Road, Boston, MA
http://www.lehoczkylab.org/
Contact: gjohnson1@g.harvard.edu

There are nearly two million Americans living with a limb amputation due either to illness or traumatic injury. Even with the best modern surgical intervention, a human limb amputation does not regenerate and results in a fibrotic scar. Amazingly, however, the amputation of a child's fingertip does not scar and will completely regenerate all of the missing tissue in the absence of surgical intervention. The medical implication of understanding the molecular basis of these two dramatically different innate responses is crucial and can potentially lead to therapeutics benefiting millions. The mouse digit tip, which similarly regenerates upon amputation, is a powerful experimental system and serves as an excellent proxy for human limb regeneration. Upon amputation, the digit tip undergoes epimorphic regeneration, a process involving the proliferation and differentiation of a mass of progenitor cells, termed the blastema, which gives rise to all of the regenerate tissue except the skin. Understanding how cells are recruited to form a blastema, how they remain proliferative, and how they organize and differentiate into appropriate tissues, are some of the key questions in regenerative biology. Our lab is focused on determining the molecular pathways and genes necessary for blastema formation, and how they coordinate proliferation and patterning of specialized progenitor cell types during digit tip regeneration. We are working towards identifying these genes and pathways and learning how to modulate them, with the ultimate goal of inducing regeneration in non-regenerative tissues.

Poster 50. Indentification of shared genetic architecture using genome-wide cross-trait analysis: novel statistical genetic method and its application in asthma and metabolic traits

Presenters: Wonil Chung, Zhaozhong Zhu
Principle Investigator: Liming Liang, lliang@hsph.harvard.edu
Harvard T.H.Chan School of Public Health
Building 2, Room 207 655 Huntington Avenue Boston, Massachusetts 02115, https://www.hsph.harvard.edu/liming-liang/
Contact: wchung@hsph.harvard.edu

Introduction: In recent years, there is a growing understanding that different diseases can have substantially shared genetic architecture. For example, recent studies have identified shared genetic variants and pathways between psychiatric disorders, asthma and allergy, lung function and chronic obstructive pulmonary diseases, sleep disturbance traits and metabolic traits, and type 2 diabetes and coronary heart diseases. However, the statistical method and pipeline for conducting such research is still limited. Methods: We introduce a novel statistical genetics approach, called genome-wide cross-trait analysis, to identify shared genetic architecture among diseases. We also developed a powerful and practical approach for multi-trait polygenic risk prediction in large cohorts, named cross-trait penalized regression (CTPR). Specifically, we propose a novel cross-trait penalty function with the Lasso and the MCP to incorporate the shared genetic effects across multiple traits. Our approach can incorporate multiple secondary traits based on individual-level genotypes and/or summary statistics from large-scale GWAS studies. Novel implementation of parallel computing algorithm makes it feasible to apply our methods to biobank-scale GWAS data. Real world application: Many clinical and epidemiological studies have shown that obesity is associated with asthma, and that associations may differ by asthma phenotype. However, genetic correlations and shared genetic components between obesity and asthma are largely unknown. We conducted a large-scale GWAS cross-trait analysis between asthma and 8 metabolic traits. We found a strong positive genetic correlation of obesity with asthma and highlighted shared immune and antigen related biological pathways between asthma and obesity. Number of hours per week: 5hrs. Requirements: Knowledge of basic genetics and statistics.

Poster 51. Early antiretroviral therapy reduces latent HIV-1 reservoirs in infants from Botswana

Presenters: Kevin Einkauf

Principle Investigator: Mathias Lichterfeld mlichterfeld@mgh.harvard.edu
Ragon Institute of MGH, MIT and Harvard
400 Technology Square, Cambridge, MA 02139,
http://www.ragoninstitute.org/portfolio-item/lichterfeld/
Contact: keinkauf@partners.org

Background: Although antiretroviral therapy (ART) can effectively suppress HIV-1 replication and improve patient outcomes, treatment discontinuation typically results in viremic rebound due to the presence of latently-infected CD4 T cells. However, early treatment during acute infection appears to limit the establishment of this viral reservoir, possibly allowing for long-term remission. The Early Infant Treatment Study in Botswana provides a unique opportunity to examine whether immediate initiation of ART can significantly decrease proviral reservoirs in HIV-1-infected infants, which may advance the search for a functional HIV-1 cure. Methods: Serial PBMC samples were collected from five infants with neonatal HIV-1 infection who started ART within 72 hours (n=4) or 31 days (n=1) after birth, and were followed for 84-96 weeks (w). Genomic DNA was subjected to near full-length amplification of single-genome HIV-1 templates. Resulting products were sequenced with Illumina MiSeq. Results: Intact full-genome proviral sequences represented an average of 41% of all detected sequences at baseline, compared to 21% of detected sequences after 84/96w of ART. This corresponded to an average frequency of 76 and 3 intact sequences per million PBMCs at baseline and after 84/96w of treatment, respectively, and is consistent with a half-life of 19 weeks for intact proviral sequences during the first two years of life. Conclusion: ART initiated very early during neonatal HIV-1 infection leads to a profound decline of intact proviral sequences in infected infants, particularly after 84/96w of treatment. Monitoring of eligible patients during future analytic treatment interruption may indicate whether long-term remission is possible.
Number of hours per week: Negotiable
Requirements: Only a willingness to learn new techniques!

Poster 52. Statistical methods for association and functional annotation of genetic variants
Presenters: Sheila Gaynor, Andy Shi
Principle Investigator: Xihong Lin, xlin@hsph.harvard.edu
Harvard Chan School of Public Health
Harvard T.H. Chan School of Public Health Building II, 4th Floor 655 Huntington Ave Boston, MA 02115,
https://content.sph.harvard.edu/xlin/index.html

Contact: sheilagaynor@hsph.harvard.edu

The Lin Lab in the Biostatistics Department works on research projects in statistical genetics and functional genomics. One primary research area aims to develop methods for mining the associations between genetic variants and multiple phenotypes in large-scale genomic consortia and biobank settings. Members of the lab have developed a number of statistical tools designed to assess association and functionality. For example, we investigate and extend principal components analysis approaches to efficiently combine information. We have applied these techniques to study the genetic basis of inflammation and immune-related risk in lung cancer. We are interested in continuing this research in other lung cancer subtypes, other diseases, and other biological systems. Other research areas aim to annotate the genome and understand the functionality of variants comprising the genetic etiology of complex diseases. For instance, we have developed methods to build tissue-specific networks from expression quantitative trait loci (eQTLs), relating genetic variants and gene expression. From such networks we are able to gain insight into the functionality of variants and integrate this insight into association analyses. We develop open source approaches to conduct genome-wide genetic and genomic analyses that are both scalable and efficient. We use high-performance computing (HPC) tools on HPC clusters. Our team includes statisticians, computational biologists, genetic epidemiologists, clinicians, and biologists, and we regularly collaborate with scientific and medical leaders. We work together within our team and with other research groups to advance scientific understanding.
Number of hours per week: Negotiable. During the academic year, 6-10 hours/week for freshmen/sophomores, 15-20 for juniors/seniors. Up to 40 hours/week in the summer for all students. Requirements: No prior research experience required. Some statistics and programming experience helpful (Stat 110, 111, 139/149; CS 50 / CS 109).

Poster 53. CRISPR/Cas9 based functional assessment of potential therapeutic approaches for USH2A associated diseases.
Presenters: Nachiket Pendse
Principle Investigator: Qin Liu, qin_liu@meei.harvard.edu
Department of Ophthalmology, Harvard Medical School, Ocular Genomics Institute, Massachusetts Eye and Ear, Broad institute
243 Charles street, Boston MA 02114,
https://oculargenomics.meei.harvard.edu/labs/pierce-lab/lab-members/
Contact: Nachiket_Pendse@meei.harvard.edu

Rationale: Mutations in USH2A are the most common cause of Usher syndrome type II, involving visual and hearing loss. It is also the most common cause of inherited retinal degenerations (IRDs) overall. Approximately 30% of USH2A cases are caused by the c.2299delG, p.Glu767fs mutation in exon 13 of the USH2A gene. Development of gene therapy for the USH2A form of arRP has been challenging, because the full length CDS of USH2A (15.6kb) far exceeds the packaging capacity of commonly used AAV viral delivery vectors for gene augmentation therapy. The USH2A gene encodes a transmembrane protein anchoring in the photoreceptor plasma membrane. There are multiple repetitive domains, including 10 laminin EGF-like domains and 35 fibronectin type 3 domains at its N terminus of USH2A. The frame-shift mutation c.2299delG, p.Glu767fs is located in exon 13 in LE domain 5. Skipping of this exon 13 (642bp) will result in an in-frame abbreviated transcript USH2A-ΔEx13. In this study, we propose to examine if the product of this abbreviated USH2A-ΔEx13 will retain partial or complete biologic function of USH2A protein. Results: We chose OC-k1 cells that are derived from mouse cochlea and express Ush2a protein and its interacting proteins at the base of the cilia under serum deprived conditions. We first created an Ush2a null cell model in OC-k1 cells using CRISPR/Cas9 technology. We demonstrate that ciliogenesis was hampered in the Ush2a null OC-k1 cells, emphasizing the important role of Ush2a protein in ciliogenesis. Moreover, our transient transfection experiments unequivocally demonstrate that the ciliogenesis in these cells can be restored using DNA plasmids expressing mouse full length (mUsh2a-FL), human full length (hUSH2A-FL) and human USH2A lacking exon 13(USH2A-ΔEx13).
Number of hours per week: 15-30 Hours per week
Requirements: Candidate should be well versed with molecular biology concepts and techniques such as genotyping PCR, Cloning etc. The candidate will get hand on experience on CRISPR based genome engineering, Cell culturing and other cutting edge molecular techniques.

Poster 54. Investigation of the role of srrA in eDNA release during biofilm formation in Staphylococcus aureus

Presenters: Carolyn Wong
Principle Investigator: Richard Losick, losick@mcb.harvard.edu
Department of Molecular and Cellular Biology, Harvard University
BL 3023 The Biological Labs 16 Divinity Ave Cambridge, MA 02138,
https://scholar.harvard.edu/rlosick/home
Contact: aksyed@g.harvard.edu

Staphylococcus aureus is a common cause of hospital-acquired infections and is of particular interest because of its ability to form biofilms, a collection of cells adhered together and to a surface. Biofilm formation enhances antibiotic resistance of S. aureus, increasing the likelihood of transmission through contaminated surfaces. Biofilms are formed when bacterial cells lyse, releasing cytoplasmic components to form a sticky matrix composed of proteins and extracellular DNA (eDNA). Additionally, during biofilm formation the pH of the environment decreases causing the proteins and eDNA to form an electrostatic net that adheres the cells together. Biofilm formation is also preceded by a decrease in the small molecule cyclic-di-adenosine monophosphate (c-di-AMP). We hypothesize that cell lysis and eDNA release are controlled by changes in c-di-AMP which regulates cell-wall integrity. This decrease in cell-wall integrity could cause cells to lyse and release the cytoplasmic components which form the biofilm matrix. Previous research from our lab suggests that the response regulator srrA contributes to eDNA release during biofilm formation. This project investigates the role of srrA in the molecular signaling pathway that leads to *S. aureus* eDNA release during biofilm formation. Further evaluation of cell-wall stability in the ΔsrrA mutant will help to elucidate the signaling network controlling S. aureus biofilm formation, which will assist in the future development of therapeutic treatments for *S. aureus* biofilm infections.

Poster 55. Nearsighted Globe: A sustainable approach to clear vision for visually impaired globally

Presenters: Mojtaba Moharrer
Principle Investigator: Gang Luo, gang_luo@meei.harvard.edu
Mass Eye and Ear, Department of Ophthalmology, Harvard Medical School, 20 Staniford Street, Boston, MA
https://www.masseyeandear.org/research/investigators/l/luo-gang

Globally there are more than one billion people with near vision impairment who can restore clear vision simply by wearing glasses. The purpose of this initiative is to address this global issue by providing a sustainable method of nearsightedness diagnosis and treatment to the patients, especially those with no or limited access to professional vision care in rural areas. Scientists at Mass Eye and Ear, Harvard Medical School launched a nonprofit initiative, Nearsighted Globe, to address the costly global health issue of myopia in developing countries. Due to a lack of eye care services and the increased cost and time associated with eye care professionals traveling to remote communities, innovative strategies need to be employed to help these communities to receive

timely and affordable eye care. NG draws together local and international stakeholders to mass produce and distribute, (1) an innovative and easy to use diagnostic instrument (refraction kit) for measuring appropriate eyeglass prescription for nearsighted patients, and (2) cost-effective glasses. Currently in the pilot phase, NG has identified sites in China, Ghana and India and will continue to identify potential communities. NG's goal is to reach 100,000 people in its first year. Currently, the team includes Optometrists, and Vision and Human Factors scientists, and Global Health specialist. We are looking for students interested in any aspects of the project including, public and global health, cultural, marketing and promotion, fundraising, quality assurance, business, finance, and law. Number of hours per week: Negotiable

Poster 56. Visualizing Ictal Networks with Granger Causality: Use with Stereotactic Electroencephalography (sEEG) to Enhance Intraoperative Decision-Making

Presenters: Joseph Madsen, MD, Eun-Hyoung Park, PhD
Principle Investigator: Joseph Madsen, MD,
Joseph.Madsen@childrens.harvard.edu
Department of Neurosurgery, Boston Children's Hospital, HMS
The Neurodynamics Laboratory Boston Children's Hospital 1 Autumn Street, 4th floor (room 403) Boston, MA 02215,
Lab page http://www.childrenshospital.org/directory/physicians/m/joseph-madsen Publications:
https://academic.oup.com/neurosurgery/article/82/1/99/3778239
Contact: Eun-Hyoung.Park@childrens.harvard.edu

Objective: Stereoelectroencephalography (sEEG) recordings are an increasingly popular means of invasively localizing the seizure onsets in patients with refractory focal epilepsy. Recently we have shown that Granger causality (GC) analysis has the potential to reveal seizure networks from interictal baseline data obtained from subdural grids. In this study, we produced "causal density" maps from interictal sEEG and again tested whether the regions of high GC would statistically resemble the topography of the seizure onset zone and resection. Methods: We analyzed interictal data from 20 consecutive patients. For each case, 10min of data was obtained immediately after all channels were connected. The "GC maps" were quantitatively compared to conventionally-constructed surgical plans and resections, by using non-parametric, rank-order statistics, as previously used in the evaluation of subdural recordings. Results: In 18 of 20 cases, the interictal GC rankings of the electrodes mapped to the seizure onset zone had higher

causality than predicted by chance (range: p<10^{-5} to 0.05). In 15 of 18 cases who had resection or ablation, causality in the resection zone was significantly increased (range: p<10^{-5} to 0.03). The aggregate probability of such a match is very small (p < 10^{-31}), suggesting that the networks highlighted in interictal GC maps correlate with surgically-relevant seizure networks. Conclusions: GC analysis applied to sEEG data has the potential to help localize ictal networks from interictal data. Since interictal data can be obtained in the operating room as the electrodes are being placed, it is possible that live GC analysis of sEEG could eventually aid surgical decision making, such as modification of the sEEG stereotactic plan to better sample highly causal regions and define the epileptogenic zone. Number of hours per week: negotiable with each student. Requirements: Our projects have number of directions that students can contribute. No prior research experience is required but if they have computer programming skills and can use computer programming language such as MATLAB (matrix laboratory) those are desirable and helpful for the projects.

Poster 57. Discovery of novel human antibodies for targeted immunotherapy

Presenters: Matthew Chang, Quan Zhu
Principle Investigator: Wayne Marasco
Wayne_Marasco@dfci.harvard.edu
Dana-Farber Cancer Institute 450 Brookline Ave Boston, MA 02215,
http://marascolab.dana-farber.org/
Contact: MatthewR_Chang@dfci.harvard.edu

Antibodies play a greater role in modern medicine than ever before and nowhere is this more apparent than in immuno-oncology. Currently, there are a number of FDA approved immune checkpoint inhibitors on the market, including pembrolizumab (Keytruda), nivolumab (Opdivo), and atezolizumab (Tenectriq) for the PD1-PDL1 axis and ipilimumab (Yervoy) for CTLA-4. While checkpoint molecules are critical in protecting healthy tissues, cancerous cells also use them to create safe havens where the tumor is invisible to the immune system. Checkpoint inhibiting antibodies are able to disrupt the tumor's defense mechanisms, allowing the patient's immune system to recognize and attack the cancerous cells. As we continue to develop a greater understanding of the biology surrounding tumors and the means by which they evade the immune system, our focus will shift from simply blocking these checkpoint receptors to antibodies that can actively modulate the microenvironment surrounding the tumor. In addition to being a viable stand-alone therapy,

these antibodies can serve as a powerful payload for genetically modified chimeric antigen receptor (CAR) T cells, allowing these engineered cells to create a favorable microenvironment that greatly increases the potency and efficacy of the host's immune system. Utilizing phage, yeast, and other display technologies, our lab seeks to discover new antibodies that can serve as a targeting moiety or novel immunomodulatory payload(s) for our CAR T cell programs. Students in the Marasco Lab will gain hands on experience in antibody discovery, characterization, and engineering and will have a unique opportunity to contribute to projects headed towards clinical development.
Number of hours per week: We recommend 10-20 hours per week, but this is negotiable and accommodations can be made.
Requirements: No prior research experience is required. Students are expected to commit to the agreed upon schedule, be well organized, have good time management skills, pay careful attention to experimental details, and have excellent communication skills. Students should also have a desire to learn and be self-motivated to carry out their projects. Completion of a general biology course is a plus.

Poster 58. Research in the Mason Group: Manipulating Entropy and Porosity in Functional Materials
Presenters: Malia Wenny
Principle Investigator: Jarad Mason, mason@chemistry.harvard.edu
Chemistry and Chemical Biology
Mallinckrodt Chemistry Laboratory 12 Oxford Street Cambridge, MA 02138, https://www.mason.chemistry.harvard.edu/
Contact: mwenny@g.harvard.edu

The Mason Group applies the tools of synthetic chemistry and nanotechnology to the design of materials that address basic science challenges in energy and sustainable development. We are particularly interested in the development of chemical strategies to manipulate entropic effects, phase transitions, and porosity at different length scales in inorganic and organic materials. Project 1: Phase-Change Materials Advanced materials that can store high capacities of thermal energy and deliver it on demand are critical to the more efficient and sustainable utilization of energy. Through the development of strategies to manipulate the thermodynamics and kinetics of order-disorder transitions, our laboratory synthesizes new phase-change materials for thermal energy storage and heat management. Project 2: Nanocrystal Frameworks Owing to their size- and shape-dependent magnetic, electronic, catalytic, optical, and mechanical properties, colloidal

nanocrystals are exceptionally powerful building blocks for the construction of tunable materials. Our laboratory investigates porous materials composed of inorganic nanocrystals bridged by rigid organic ligands. These materials will exhibit novel properties that are derived from individual nanocrystals and their collective interactions, along with functionalized organic ligands and guest species in well-defined pores. Project 3: Porous Liquids Liquids present a new and unexplored prospect for porous materials, as liquids posses useful properties not found in solids. Our laboratory is interested in the development of both liquids with intrinsic porosity and nanocrystal-based porous liquids. These materials offer the opportunity to address long-standing challenges in adsorption as well as to develop new self-cooling and self-heating adsorbents and membranes for water purification.
Number of hours per week: Negotiable, but prefer students who can work at least 10-15 hours/week
Requirements: No prior experience required, but prefer students who have taken at least one lab course and have taken (or plan to take) CHEM 40

Poster 59. Mice, motor control and machine learning
Presenters: Gary Kane, Tanmay Nath
Principle Investigator: Mackenzie Mathis, mathis@rowland.harvard.edu
The Rowland Institute at Harvard
100 Edwin H. Land Blvd, mousemotorlab.org

The adaptive motor control lab is looking for undergraduate research assistants to help with research on how the brain controls movement. Our lab conducts experiments on mice where we ask them to adapt their movements to the environment. For example, we utilize a paradigm in which we teach them to use joysticks to collect rewards. We use state-of-the-art techniques to record and manipulate neural activity across many brain regions (e.g. two-photon microscopy, single-unit physiology, and optogenetics) and machine learning techniques to track animal movement, to analyze complex neural data, and to build computational models of neural activity and behavior. Read more about our work here: www.mousemotorlab.org . Number of hours per week: Typically 10 hours/week during the semester, and full time during the summer months. Terms are negotiable, and depends on the arrangement with each individual student. Requirements: Prior experience with animals and Python programming are highly desirable.

Poster 60. Effects of changes in climate and land use on wildfire smoke and dust particulate matter in the western US

Presenters: Yang Li
Principle Investigator: Loretta Mickley, mickley@fas.harvard.edu
Harvard John A. Paulson School of Engineering and Applied Sciences
29 Oxford St, Cambridge, MA 02138,
https://www.seas.harvard.edu/directory/mickley
Contact: yangli@seas.harvard.edu

Particulate matter (PM) has adverse consequences for human health and visibility. Previous studies have projected increasing trends of wildfire smoke and dust PM under a warmer and drier climate in the western US. However, there exists large uncertainty in these projections. Here we use the LPJ-LMfire model, a process-based fire model linked to a dynamic global vegetation model, to assess the impacts of future changes in climate and land use practices on wildfires and land cover in the mid- and late- 21st century for different Representative Concentration Pathways (RCPs). We then apply the future wildfire emissions and land cover from LPJ-LMfire to GEOS-Chem, a global chemical transport model. In this way, we can quantify future wildfire smoke and dust mobilization and transport and assess the consequences for surface air quality for both moderate (RCP4.5) and worst-case (RCP8.5) climate scenarios. The distribution of wildfire smoke and dust PM can then be linked to human exposure at the city scale. Our study will provide valuable resources for environmental managers to better prepare for the air quality challenges in a changing world.
Number of hours per week: 6-10 hours/week and negotiable.
Requirements: Interest in programming is essential, and some knowledge of statistics would be helpful.

Poster 61. Effects of aging and tendon anatomical location on tendon structure and tendon stem/progenitor cell (TSPC) properties

Presenters: Ege Eskibozkurt
Principle Investigator: David Mooney, mooneyd@seas.harvard.edu
Postdoctoral Fellow at Mooney Lab
Room 415, Engineering Sciences Lab 58 Oxford St. Cambridge, MA 02138, https://mooneylab.seas.harvard.edu/
Contact: bfreedman@seas.harvard.edu

Musculoskeletal injuries affect 33 million people in the United States per year and 50% of these injuries involve tendons and ligaments. Although aging may contribute to tendon degeneration and injury, these effects

depend on the particular tendon type. The effects of aging on tendon structure and tendon stem/progenitor cells (TSPCs) from different anatomical locations (e.g., shoulder, knee, and ankle) has not been elucidated previously. Therefore, the purpose of this study was to identify how aging and anatomical location affect tendon morphology and TSPC properties. Tendons from different anatomical locations (Achilles (AT), patellar (PT), flexor digitorum longus (FDL) and rotator cuff tendons (RC)) were harvested (n=6/group). High frequency ultrasound imaging was used to assess morphological and structural differences across different tendon types and across different ages. TSPCs isolated from tendons were cultured and prepared for flow cytometry to screen for markers of TSPCs (CD90, CD146 and CD44). A scratch assay was performed to assess cell migration. Patellar tendons were thicker and exhibited a more elliptical shape and thickness compared to AT and FDL. Cross sectional area was increased in aged AT compared to the young animals. Current work is evaluating how aging and anatomical location affect TSPC behavior in vitro. Taken together, this work helps identify the effect of aging and anatomical location on TSPCs and provides insight into therapeutic applications of these TSPC populations to augment tendon healing.
Number of hours per week:

Poster 62. Role of T Cells in the Autoimmune Disease Systemic Lupus Erythematosus

Presenters: Melissa Carr-Reynolds, Takayuki Katsuyama
Principle Investigator: Vaishali Moulton, vmoulton@bidmc.harvard.edu
Division of Rheumatology, Department of Medicine Beth Israel Deaconess Medical Center, Harvard Medical School
3 Blackfan Circle, CLS-928, Boston MA 02215,

Autoimmune diseases occur when the immune system fails to recognize “self” and attacks the body’s healthy tissues. Systemic Lupus Erythematosus (SLE) or lupus, is a debilitating chronic multi-system autoimmune disease with no cure, that predominantly affects women in the childbearing years. Painful joints and skin lesions are frequent manifestations, while complications can lead to kidney failure. Genetics, environmental factors and hormones are implicated in lupus pathogenesis. Abnormal T cells contribute to lupus pathogenesis. Our goal is to understand molecular defects, which contribute to T cell dysfunction in lupus. Our research has identified a new molecule serine arginine-rich splicing factor 1 (SRSF1), which controls genes important for T cell function. SRSF1 is decreased in lupus patients especially in

patients with severe disease. We have generated mice, which lack Srsf1 selectively in T cells, and these mice develop lupus-like disease. Ongoing research in the lab is focused on understanding the mechanisms related to the role and regulation of SRSF1 in T cells and autoimmune disease in mice and humans. One project studies the role of the female hormone estrogen in the regulation of SRSF1 in human peripheral blood T cells from healthy donors and lupus patients. Other projects include animal studies in Srsf1-knockout mice and lupus-prone mice to understand how SRSF1 controls T cell physiology and function and its role in autoimmune disease. In summary, our lab conducts basic and translational biomedical research utilizing cellular immunology, cell biology, biochemistry and molecular biology approaches, with the overall aim of better understanding a complex disease to pave the path to identify better drug targets and biomarkers.
Number of hours per week: 15-20 hours/week. 40 hours/week in the Summer. Requirements: No prior research experience is required. However some research experience is preferable - such as part time or summer research work in a biological life sciences lab.

Poster 63. Brachial Plexus Block with Liposomal Bupivacaine (EXPAREL) Compared to Placebo Showed Improved Analgesia and Reduced Opioid Consumption after Shoulder Surgery

Presenters: Xiang Bai
Principle Investigator: Srdjan Nedeljkovic, snedeljkovic@partners.org
Department of Anesthesiology, Perioperative and Pain Medicine, Brigham and Women's Hospital
850 Boylston Street, Chestnut Hill, MA 02476
https://www.brighamandwomens.org/anesthesiology-and-pain-medicine/Research/srdjan-nedeljkovic-research
Contact: xbai@bwh.harvard.edu

In treating postoperative pain after major shoulder surgery, commonly used postoperative approaches like single-injection of analgesics are limited by their short duration of analgesia and continuous peripheral nerve blocks are associated with catheter-related complications. Therefore, there is a clinical need for single-dose local anesthetics that can provide prolonged analgesia after this type of major surgery. This double-blind, multicenter Phase 3 study evaluated the efficacy, safety, and pharmacokinetics of single-injection of EXPAREL, liposomal bupivacaine, in brachial plexus block for shoulder surgery. Adult patients (n=140) scheduled for primary unilateral total shoulder arthroplasty or rotator cuff repair were randomized 1:1 to receive a single-injection of

EXPAREL 133 mg or placebo (EXPAREL 133 mg, n=69; placebo, n=71) by ultrasound-guided brachial plexus block. The area under curve of pain scores, opioid consumption, median time to opioid rescue, and percentage of opioid-free patients through 48 hours were significantly improved in the EXPAREL group in comparison with the placebo group. There was no difference in incidence of adverse events between the groups. In conclusion, brachial plexus block with single-injection of 133 mg EXPAREL was safe and well tolerated in shoulder surgery patients and demonstrated improved analgesia with reduced opioid use.
Number of hours per week: It is negotiable, and depends on arrangement with each individual student
Requirements: Great passion for clinical research and having positive impact on patients' life Absolutely integrity and responsibility to conduct research Biological or biomedical background

Poster 64. Beyond Mapping the Brain's Connections: Novel Computational Analyses and Clinical Applications
Presenters: Lauren O'Donnell
Principle Investigator: Lauren O'Donnell, odonnell@bwh.harvard.edu
Harvard Medical School
1249 Boylston St. Boston, MA
https://scholar.harvard.edu/laurenjodonnell
Contact: odonnell@bwh.harvard.edu

Our research focuses on diffusion magnetic resonance imaging, the only method that can map the connections of the living human brain. We have multiple related research directions: development of novel computational methods for analysis of brain MRI, application of these methods to study the brain in health and disease, robust analysis of large clinical datasets, creation of open-source software, and improvement of brain mapping for neurosurgical planning. Opportunities exist for students to help with research projects in all of these areas. Dedicated students with sufficient experience in the lab have the opportunity to conduct a research project. Current lab members and visitors include both technical and clinical researchers. For videos of our recently released white matter brain atlas (created with a combination of machine learning and expert neuroanatomical knowledge), please see: http://dmri.slicer.org/atlases/
For recent examples of our research and software, please see:
https://www.sciencedirect.com/science/article/pii/S1053811918305342
http://cancerres.aacrjournals.org/content/77/21/e101.short
Number of hours per week: negotiable

Requirements: No prior research experience is required. Depending on their interests, students will learn about python programming, neuroanatomy, statistical analyses, and/or other aspects of our research.

Poster 65. Relationships of sleep with resting state functional connectivity in trauma exposed individuals with and without PTSD: The Sleep and Anxiety Disorders Laboratory, MGH/HMS Department of Psychiatry

Presenters: Jeehye Seo, Ph.D., Edward F. Pace-Schott, Ph.D.
Principle Investigator: Edward F. Pace-Schott, Ph.D., epace-schott@mgh.harvard.edu
Harvard Medical School Massachusetts General Hospital
Massachusetts General Hospital - East CNY 149 13th Street Room 2605
Charlestown, MA 02129
https://sleep.med.harvard.edu/people/faculty/255/Edward+Franz+Pace-Schott+M+S+M+A+Ph+D
Contact: amoir710@gmail.com

The Sleep and Anxiety Disorders Laboratory is part of the MGH/HMS Department of Psychiatry. We use polysomnography, fMRI, psychophysiology, actigraphy, self-report and related techniques to investigate how sleep helps humans regulate emotions. Specifically, we study the influence of sleep disturbance on the development of anxiety and traumatic-stress related disorders. Sleep plays an important role in the adaptive consolidation of emotional memory. On the other hand, sleep disorders increase the odds of incident anxiety disorders and of PTSD following a traumatic stressor. We are interested in how sleep influences evolutionarily ancient learning and memory processes that are deficient in anxiety disorders and PTSD. Such processes include fear extinction--learning that a once feared object or event is no longer dangerous--and habituation whereby we become less reactive to frequently encountered stimuli. We study effects of sleep on these elemental forms of emotional memory at the behavioral, physiological and neural level using experimental models of extinction and habituation. We also examine sleep effects on the primary psychotherapeutic application of extinction learning–exposure therapy–a first-line treatment for anxiety disorders and PTSD. We currently conduct two NIMH-funded projects. The first examines interrelationships between hyperarousal, sleep quality and memory for the extinction of conditioned fear among persons who have experienced a recent traumatic event. The second tests the hypothesis that insomnia in Generalized Anxiety Disorder worsens fear extinction and disrupts functional connectivity between fear

expression and emotion regulatory structures. Lab volunteers will learn about sleep science, anxiety disorders, physiological signs of emotion, sleep physiology and structural and functional MRI.
Number of hours per week: 10-15 h/wk. This is negotiable depending on the student's goals (e.g., senior thesis vs. obtaining lab experience)
Requirements: Requirements are to have: (1) at least Sophomore status, (2) a psychology or neuroscience academic focus, (3) a strong interest in behavioral neuroscience, sleep science or psychiatry/abnormal psychology, and (4) a willingness to devote 10 hours per week to work at or for the lab.

Poster 66. CMOS nano-electrode arrays for high-throughput electrophysiology
Presenters: Tianyang Ye, Jeffrey Abbott
Principle Investigator: Hongkun Park, hongkun_park@harvard.edu
School of Engineering and Applied Sciences
12 Oxford St, Cambridge, MA, https://hongkunparklab.com/
Contact: tianyangye@g.harvard.edu

The paralellization of intracellular recording can greatly benefit the study of complex neural networks, but it has proven to be hard to achieve. In this work, we combine the intracellular recording capabilities of nano-electrodes with the massive parallelism of complementary-metal-oxide-semiconductor (CMOS) circuit to build the CMOS nano-electrode array (CNEA) device. Our experiments demonstrate the device's capability to record from hundreds to thousands of cardiomyocytes to neurons within cardiac tissues or neural networks.
Number of hours per week: ~10 hrs. Requirements: no requirements

Poster 67. Ionizing radiation causes collective migration of the airway epithelium
Presenters: Michael O'Sullivan, Jennifer Mitchel
Principle Investigator: Jin-Ah Park, jpark@hsph.harvard.edu
Harvard TH Chan School of Public Health
Building 1, Room 306 665 Huntington avenue, Boston, MA 02115,
https://www.hsph.harvard.edu/park-lab/
Contact: mosullivan@hsph.harvard.edu

In the lung, airway epithelial cells are the first line of defense against external stimuli. Either excessive stimuli or diseased states cause the disruption of epithelial integrity. Using an in vitro compression model that mimics asthmatic bronchospasm, we discovered a striking migratory

phenotype of airway epithelial cells (Park et al., Nat Mater. 2015). When airway epithelial cells are mature, the cells are immobile and jammed. However, when cells are exposed to perturbation mimicking asthmatic bronchospasm, these jammed cells become mobile and unjammed, which is termed an unjamming transition. During the unjamming transition, cell shape changes from a cobblestone-like shape to an elongated shape. Furthermore, a sustained unjammed phase is associated with asthma. Our unpublished data show that rhinovirus infection also provoked unjamming of the cells. However, mechanisms underlying the unjamming transition remain unknown. Thus, we hypothesize that epithelial cell injury promotes collective migration of the cells by the unjamming transition. To induce epithelial cell injury, we utilized ionizing radiation that causes double-strand DNA beaks. Primary human airway epithelial cells were cultured in an air-liquid interface. Then, well-differentiated airway epithelial cells were exposed to ionizing x-rays. Unjamming was determined by two well-characterized features: increased cell motility and elongated cell shape. Our data indicate that ionizing radiation causes collective migration of epithelial cells, which depends partially on TGF-β receptor pathway. This supports the idea that injured epithelium exhibits a distinct physical phenotype, which is known as unjamming. Further investigation into the unjammed epithelial layer is required for a comprehensive understanding of injured epithelium and its role in lung disease.
Number of hours per week: Prefer to up 40hrs/wk during summer, but it is negotiable. It depends on arrangement with each individual student.
Requirements: Strong motivation and excitement for Science!

Poster 68. Goal Recognition Design for Enhanced Human-Robot Interaction

Presenters: Sarah Keren
Principle Investigator: David Parkes, parkes@eecs.harvard.edu
Center for Research on Computation and Society (CRCS) John A. Paulson School Of Engineering And Applied Sciences Harvard
29 Oxford St, Cambridge, MA 02138, https://econcs.seas.harvard.edu/; http://sarahkeren.wixsite.com/sarahkeren-academics/goal-recognition-design
Contact: skeren@seas.harvard.edu

Goal recognition design (GRD) is the task of redesigning environments in order to facilitate online goal recognition. As such, while goal recognition tools are typically aimed at efficiently analyzing online observations of agents (i.e., users - human or automated) in order to infer their

objective, GRD focuses on manipulating the environment in which agents act to guarantee early recognition. In a nutshell, given a model of a domain and a set of possible goals, a solution to a GRD problem determines: (1) to what extent do actions, performed by an agent within the model, reveal the agent's objective? and (2) what is the best way to modify the model so that the objective of an agent is revealed as early as possible? GRD answers these questions by offering a solution for assessing and minimizing the maximal progress of an agent in the model before recognition is guaranteed. This approach is relevant to any domain for which quickly performing goal recognition is essential and in which the model design can be controlled. Applications include intrusion detection, assisted cognition, computer games, and many more. Our current focus is on applying the GRD framework to a human-robot collaboration setting, where goal recognition is done by a robot in order to assist a human user accomplish her tasks. This requires both implementing the GRD ideas within this new automated setting, and accounting for different forms of uncertainty inherent in the problem description. In particular, in order to enhance the robot's performance, our design process needs to account for the limited perception and knowledge of both the robot and human user. This offers a new and exciting opportunity to extend our framework and broaden its applicability to many real world setting to which GRD can be applied. Number of hours per week: Negotiable. Requirements: Some programming skills are preferable.

Poster 69. Aggregated single-study learners for generalizable predictions

Presenters: Boyu Ren
Principle Investigator: Giovanni Parmigiani, gp@jimmy.harvard.edu
Dana-Farber Cancer Institute Harvard T.H. Chan School of Public Health
3 Blackfan Circle, Boston, MA 02115
https://scholar.harvard.edu/parmigiani
Contact: bor158@mail.harvard.edu

Replicability of the performance of prediction models across different studies gains increasing attention in the scientific community. Recently, a method based on ensemble learning is proposed to address this problem. This method utilizes stacking in machine learning to create a consensus prediction model based on ensembles of prediction models trained on different studies. When study-to-study heterogeneity is large, the consensus model has smaller out-of-study prediction errors comparing to pooling all studies and training a single model based on the

merged data. In this paper, we propose a theoretical framework to justify the optimality of the consensus model in terms of performance replicability. The framework further alludes to a more principled approach for combining ensembles of prediction models that overcomes potential over-fitting. We also explore the visualization of study heterogeneity via the consensus prediction model, which provides crucial information about the reliability of the consensus model in a given subset of a covariate space. We apply this our approach to an air pollution dataset to predict mortality rate based on air quality. We show that our predictor is robust against the geographical heterogeneity of the effects of pollutants and has better prediction accuracy than city-specific prediction models by borrowing information across cities.

Poster 70. You Are What You and Your Parents Ate During Early Life! - Impact of Early Life Nutrition and Metabolism on Epigenetic Regulation and Risk for Type 2 Diabetes and Obesity during Adult Life

Presenters: Soravis Osataphan
Principle Investigator: Mary Elizabeth Patti
mary.elizabeth.patti@joslin.harvard.edu
Joslin Diabetes Center, 1 Joslin Place
https://joslinresearch.org/investigators/Mary-Elizabeth-Patti
Contact: soravis.osataphan@joslin.harvard.edu

The overall goal of research in the Patti Lab at the Joslin Diabetes Center (Longwood area) is to identify the molecular mechanisms mediating type 2 diabetes risk, particularly those arising at the gene-environment interface, which alter epigenetic regulation of gene expression. We are particularly interested in how early life exposures to suboptimal nutrition or metabolism can “program” increased disease risk in offspring during later life. Our laboratory utilizes cellular and animal models to study how these early life exposures impact the function of stem cells and tissues critical for metabolism. We have recently discovered that nutrition and metabolism in either male or female parents can also influence health of subsequent generations, via epigenetic modifications in germ cells. This in turn suggests that improving health of parents before conception may be a strategy to reduce obesity and diabetes in their children. Related clinical/translational studies are focused on understanding how interventions to improve the health of fathers before conception can influence epigenetic marks in sperm, and thus early post-fertilization development and child health. In related studies, we investigate mechanisms by which bariatric surgery causes

remission of type 2 diabetes, and collaborate with Harvard engineering (SEAS) to develop novel approaches to treatment of post-bariatric hypoglycemia (e.g. closed-loop glucagon delivery systems). Number of hours per week: negotiable. Requirements: no prior skills required.

Poster 71. Major evolutionary transitions in the vertebrate fossil record

Presenters: Katrina Jones, Zachary Morris
Principle Investigator: Stephanie Pierce, spierce@oeb.harvard.edu
Department of Organismic and Evolutionary Biology Museum of Comparative Zoology Harvard University, 26 Oxford St, Cambridge, MA
https://projects.iq.harvard.edu/spierce
Contact: katrinajones@fas.harvard.edu

The Pierce lab uses the fossil record to piece together major evolutionary transitions in vertebrate animals. We study the evolutionary history of a broad range of vertebrate groups (fish, amphibians, reptiles, birds, and mammals) through careful examination of living and fossil anatomy using cutting-edge techniques such as: CT scanning, biomechanical experimentation, and computer modelling. We are currently looking for undergraduates to get involved with a range of projects in the lab. Mammal backbones - The mammal backbone is specialized into distinctive anatomical regions, which helps mammals to move in an amazing variety of ways. We are studying vertebral biomechanics in living mammals and understanding the function of backbones in the extinct mammal forerunners using computer modelling. Crocodile skull development - The skull of crocodiles plays a role in nearly all ecological interactions; however, its function constantly changes as individuals develop from embryos tojuveniles to adults. We are studying how the crocodile skull has evolved over millions of years and how development has driven or facilitated this evolution. Tetrapod limb evolution - The transition from water-to-land, and the associated evolution of tetrapods limbs, was one of the most important evolutionary events in the fossil record. We are studying how limbs evolved to meet the new functional requirements of terrestrial locomotion by analyzing both how shape and functional traits changed across water-land transition. If you are curious about vertebrate evolution, or want to develop skills in comparative evolutionary anatomy, biomechanics, imaging or computational methods, please come and speak to us!
Number of hours per week: Negotiable
Requirements: No requirements

Poster 72. Center for depression, anxiety and stress research
Presenters: Amelia Moser
Principle Investigator: Diego Pizzagalli, dap@mclean.harvard.edu
McLean Hospital
115 Mill St., Belmont, MA 02478, https://cdasr.mclean.harvard.edu/
Contact: djcrowley@mclean.harvard.edu

The Center for Depression, Anxiety and Stress Research (CDASR; Director: Diego A. Pizzagalli, Ph.D.) was launched in 2010 at McLean Hospital - the largest psychiatric facility of Harvard Medical School. Using an interdisciplinary approach, CDASR investigators are working to identify the biological, environmental, and psychological factors that contribute to depression and anxiety. The ultimate goal of this research is to develop better prevention and treatment strategies for these prevalent disorders. The Center is located in the deMarneffe Building at McLean Hospital. Combining outstanding research facilities with world-class clinical care, McLean and CDASR provide a unique mix of cutting-edge research focused on depression and anxiety.
Number of hours per week: 10-16 hours per week
Requirements: 10-16 hours per week required. Some prior research experience preferred.

Poster 73. Cardiac gene regulation, regeneration, and disease modeling.
Presenters: Rachelle Ambroise, Jardin Blake
Principle Investigator: William Pu, wpu@pulab.org
Boston Children's Hospital and Harvard Medical School
Enders Research Building, Boston Children's Hospital, 300 Longwood Ave, Boston, MA 02115, www.pulab.org
Contact: wpu@pulab.org

The Pu lab is broadly interested in (1) cardiac gene regulation, primarily at the transcriptional level; (2) cardiac regeneration; and (3) cardiac disease modeling using iPSC and mouse models. Our work spans basic studies of transcriptional and epigenetic gene regulation to translational studies of gene therapy for heart disease. We are looking for energetic, motivated students with interest in cardiac biology, gene regulation, stem cells, developmental biology, regenerative medicine, gene therapy, or bioinformatics. We use genetic, biochemical, and computational approaches in stem cells and mouse models. Here, we describe a project about the regulation of cardiomyocyte maturation. After birth, cardiomyocytes (CM) acquire numerous adaptations in order to

efficiently pump blood throughout an animal's lifespan. How this maturation process is regulated and coordinated is poorly understood. Here, we perform a CRISPR/Cas9 screen in mice and identify serum response factor (SRF) as a key regulator of CM maturation. Mosaic SRF depletion in neonatal CMs disrupts many aspects of their maturation, including sarcomere expansion, mitochondrial biogenesis, transverse-tubule formation, and cellular hypertrophy. Maintenance of maturity in adult CMs is less dependent on SRF. This stage-specific activity is associated with developmentally regulated SRF chromatin occupancy and transcriptional regulation. SRF directly activates genes that regulate sarcomere assembly and mitochondrial dynamics. Perturbation of sarcomere assembly but not mitochondrial dynamics recapitulates SRF knockout phenotypes. SRF overexpression also perturbs CM maturation. Together, these data indicate that carefully balanced SRF activity is essential to promote CM maturation through a hierarchy of cellular processes orchestrated by sarcomere assembly.
Number of hours per week: 10-20 hrs/wk, summers encouraged; depends on each student's background and situation.
Requirements: Enthusiastic attitude and interest in gene regulation, cardiac biology, or bioinformatics. Prior research experience is not required, but those without prior experience need greater commitment and a longer time horizon.

Poster 74. The Biomechanics of Hearing
Presenters: Hamid Motallebzadeh, Nam-Hyuan Cho
Principle Investigator: Sunil Puria, Sunil_Puria@meei.harvard.edu
Harvard Medical School Mass. Eye and Ear
Eaton-Peabody Laboratory, 243 Charles Street, Boston, MA, 02114, UA,
https://projects.iq.harvard.edu/otobiomechanics
Contact: hamid_motallebzadeh@meei.harvard.edu

We study the biomechanics of the ear—one of the world's most extraordinary biological sensors. Research into the auditory system brings together fields as diverse as engineering, computational modeling, signal processing, medicine, audiology, physiology, psychoacoustics, neuroscience, and imaging. As a result of this breadth, students can become proficient in a variety of distinct disciplines, techniques, and research approaches, while also developing interdisciplinary problem-solving skills. Our current projects involve the scientific, engineering, and clinical aspects of cochlear and middle-ear biomechanics, hearing-loss prevention in high-noise environments, devices that deliver sound through the bone-conduction pathway, and the commercial development

of novel hearing aids that directly actuate the middle ear. These studies incorporate physiological experiments for determining structure–function relationships within the ear; imaging studies for obtaining the 3-D anatomy of the tiny structures within the middle ears and cochleae of multiple species; and the development of computational models that incorporate the imaging results, can be validated against the physiological results, and can be used to test hypotheses related to clinical and technological improvements for the treatment and prevention of hearing loss. https://projects.iq.harvard.edu/otobiomechanics.
Number of hours per week: 8 to 15 hours/week.
Requirements: Curiosity and good quantitative skills.

Poster 75. TSC1 Patient NPCs reveal altered early neurodevelopmental phenotypes and mTORC1-independent signaling defects

Presenters: Pauline Martin
Principle Investigator: Vijaya Ramesh, ramesh@helix.mgh.harvard.edu
Massachusetts General Hospital, Boston
Center for Genomic Medicine Department of Neurology, Massachusetts General Hospital 185 Cambridge Street, Simches Building 02114 Boston, https://cgm.massgeneral.org/
Contact: pmartin12@mgh.harvard.edu

Tuberous sclerosis complex (TSC) is a multisystem neurodevelopmental disorder that includes epilepsy, autism spectrum disorder (ASD), intellectual disability (ID), as well as tumors in many organs. TSC is caused by mutations in the TSC1 or TSC2 gene, and the TSC1/2 protein complex inhibit mTORC1 signaling pathway that control cell size, proliferation and protein translation. The aberrant activation of mTORC1 in TSC has led to treatment with mTORC1 inhibitor rapamycin analogs as a lifelong therapy for the TSC-associated tumors, but the neurocognitive symptoms remain unaffected. Here, we generated patient-specific induced pluripotent stem cells (iPSCs) from a TSC1 patient and established genetically matched isogenic set lines of Heterozygous (het), Null and Corrected Wild Type (Corr-WT) iPSCs using CRISPR/Cas9. We then differentiated these lines into Neural Progenitor Cells (NPCs). TSC1-Het and Null NPCs revealed increased proliferation and altered neurite outgrowth, when compared with the matched WT. Recent studies suggest that earlier developmental events such as NPC proliferation, and neurite outgrowth that occur prior to neuronal differentiation are critical in disease pathogenesis of neurodevelopmental disorders such as autism. TSC1-Het and Null NPCs

also displayed a basal activation of ERK1/2, which was further activated upon rapamycin treatment. Rapamycin also increased MNK1/2-eIF4e signaling in NPCs. Because MNK-eIF4E and MEK-ERK are known to regulate protein translation, our results suggest that these pathways could play a role in the CNS manifestations of TSC. Therefore combining a MEK or MNK inhibitor with rapmaycin may be more beneficial for TSC-associated CNS defects. We have performed transcriptome analyses to identify differentially expressed genes and networks that could reveal novel therapeutic targets. Number of hours per week: negotiable. Requirements: no prior research experience required.

Poster 76. Magnetic Resonance Spectroscopy of Brain Tumors

Presenters: Eva-Maria Ratai
Principle Investigator: Eva-Maria Ratai, eratai@mgh.harvard.edu
Massachusetts General Hospital
149 13th Street, Charlestown, MA 02129,
https://nmr.mgh.harvard.edu/lab/ratailab

We are looking for Harvard undergraduate students in Life Sciences who are interested in conducting research related to Neuroimaging. Glioblastomas (GBM) are challenging cancers to treat, and positive clinical outcome in patients with recurrent glioblastoma multiforme continues to be low. One of the most informative imaging tools to monitor treatment response or treatment failure in brain tumors such as GBM is magnetic resonance imaging (MRI). MRI is a non-invasive technique primarily used in medical settings to produce high quality images of the inside of the human body. In addition, magnetic resonance spectroscopy (MRS) is a promising imaging technique that enables investigators to determine the presence and amount of specific metabolites. Thus, MRS provides information about the metabolic activity of tumors, and may give physicians critical insight into tumor activity. The student will work under direct supervision of Dr. Eva-Maria Ratai who holds the title of Clinical Spectroscopist at MGH for 15 years. Furthermore, the candidate will work directly with MGH radiologists and neurologists on this project. This work will provide the candidate with research experience in neuroimaging, may lead to a conference abstract and papers, and will aid in her/his future career as neuroscientist or physician. Number of hours per week: Negotiable.
Requirements: No prior research experience is required.

Poster 77. Huntington's disease pattern of transcriptional dysregulation in the absence of mutant huntingtin is produced by knockout of neuronal GLT-1

Presenters: Paul Rosenberg
Principle Investigator: Paul Rosenberg
paul.rosenberg@childrens.harvard.edu
Boston Children's Hospital Harvard Medical School
Center for Life Science 3 Blackfan Circle Boston, MA 02115,
http://brain.harvard.edu/?people=paul-rosenberg

Chronic neurodegenerative diseases (CNDs) are all thought to cause a synaptic dysfucntion leading to synaptic removal by the innate immune system. The cause of synaptic dysfunction in any CND is unknown. GLT-1 is the major glutamate transporter in the brain, and is expressed in astrocytes and in axon terminals in the hippocampus, cortex, and striatum. Neuronal GLT-1 accounts for only 5-10% of total brain GLT-1 protein, and its function in axon terminals is uncertain. We have generated a conditional GLT-1 knockout to study the function of GLT-1expressed in neurons. In HD, synaptic dysfunction of the corticostriate synapse is well-established. Transcriptional dysregulation is a key feature of HD. We hypothesized that deletion of neuronal GLT-1, because it is expressed in axon terminals in the striatum, might produce a synaptopathy similar to that present in HD. If true, then some of the gene expression changes observed in HD might also be observed in the neuronal GLT-1 knockout. In situ hybridization using 33P labeled oligonucleotide probes was carried out to assess localization and expression of a panel of genes known to be altered in expression in HD. We found changes in the expression of four out of eight genes chosen that are known to be altered in expression in HD. The changes in expression in the neuronal GLT-1 knockout of genes thought to exemplify HD transcriptional dysregulation suggest an overlap in the synaptopathy caused by neuronal GLT-1 KO with the synaptopathy caused by HD. Understanding the synaptic abnormality in the neuronal GLT-1 KO may help elucidate the basis for the synaptic abnormaility in HD and, possibly, other CNDs. Number of hours per week: Negotiable Requirements: Prior research experience is desirable but not necessary.

Poster 78. Radiation Imaging Instrumentation

Presenters: Salar Sajedi, Lisa Blackberg
Principle Investigator: Hamid Sabet, hsabet@mgh.harvard.edu
149 13th St, Rm# 5213
Contact: hsabet@mgh.harvard.edu

In Sabet-lab for radiation imaging instrumentation, we focus on developing high-performance and advanced radiation imaging systems by rigorously studying and addressing fundamental limitations of today's imaging systems. One specific research area in our lab is fabrication of new category of radiation detectors using ultra short laser pulses for Positron Emission Tomography (PET), Single Photon Emission Computed Tomography (SPECT), Computed Tomography (CT) applications, and intraoperative nuclear probes. We developed Laser-Induced Optical Barriers (LIOB) technique where a high-power pulsed laser is tightly focused inside the bulk of a radiation scintillator crystal, causing a local modification of the crystal structure. The modification is manifested by a change in refractive index (RI) of the material, where the modified region (called Optical Barrier) will have a RI lower than that of the unmodified crystal. Using the LIOB technique we place a pattern of optical barriers to manipulate scintillation light spread inside the radiation detector to achieve high spatial resolution radiation detector and hence improve the medical image quality. Our ongoing projects include development of brain-dedicated PET detector system, development of prototype sub-mm resolution small-animal PET, design and optimization of a cardiac-specific SPECT system, development of a scintillator-based photon counting CT detector, and fabrication of intra-operative positron-gamma probe for surgical application. These multidisciplinary projects span over medical and nuclear physics, engineering, mathematics, computer modeling, and material science. Number of hours per week: flexible, depends on arrangement with each individual student
Requirements: Knowledge MATLAB, Python, C, GPU programing, or any area of physics, electronics, optics, and laser is a plus.

Poster 79. Metabolic regulation of retinal epithelial cell phenotype and antioxidant capacity: implications for retinal degenerative diseases

Presenters: Mariana Rosales, Sangeeta Satish
Principle Investigator: Magali Saint-Geniez
magali_saintgeniez@meei.harvard.edu
Harvard Medical School Schepens Eye Research Institute of Massachusett Eye and Ear
Schepens Eye Research Institute 20 Staniford Street Boston, MA 02114, https://www.masseyeandear.org/research/investigators/s/saint-geniez-magali

Purpose: Retinal pigment epithelium (RPE) dysfunction due to oxidative damage and epithelial-to-mesenchymal transition (EMT) is a key event in

severe ocular diseases such as age-related macular degeneration (AMD) and retinal vitreoretinopathy (PVR). Our group has shown that PGC-1α, a critical regulator of mitochondrial function, promotes RPE oxidative metabolism and antioxidant capacity. Here, we examined the consequences of PGC-1α loss in vitro and in vivo and tested the therapeutic potential of ZLN005, a selective PGC-1α activator, in protecting RPE from cytotoxic oxidative damage. Method: PGC-1α was silenced in ARPE-19 by lentiviral delivery of shRNA (shPGC-1a). Non-silencing shRNA was used as control (shControl). Cells were maturated and their metabolism, autophagic flux and phenotype evaluated. RPE-specific PGC-1s KOs were generated by subretinal injection of AAV2/1-Cre in adult C57Bl/6J;α,β floxed mice. Following AAV-delivery, morphological and functional analysis was conducted by fundus photography, OCT, ERG and TEM. ZLN005 cytoprotective functions were tested on parental ARPE-19 cells exposed to the prooxidants H2O2 and NaIO3. Results: PGC-1α deficiency in RPE cells affected mitochondria/autophagy function ultimately inducing EMT. Adult conditional knockout of PGC-1 coactivators resulted in rapid RPE dysfunction and severe photoreceptor degeneration comparable to the ones observed in AMD and PVR. Treatment with ZLN005 protected RPE cells from oxidative damage through selective induction of PGC-1α and target anti-oxidant enzymes. Conclusion: These findings show that PGC-1α is required to maintain RPE functional and phenotypic status by promoting oxidative metabolism and autophagy-mediated repression of EMT. ZLN005 may serve as a novel therapeutic agent for retinal diseases associated with RPE dystrophies.
Number of hours per week: Weekly hours are negotiable
Requirements: Prior research experience is not required but we are looking for highly motivated individuals. Basic laboratory skills and cell culture experience is a plus.

Poster 80. Continuous EEG monitoring in extracorporeal membrane oxygenation (ECMO) in the pediatric and neonatal intensive care unit

Presenters: Melissa DiBacco
Principle Investigator: Arnold Sansevere
arnold.sansevere@childrens.harvard.efu
Neurology, Boston Children's Hospital Harvard Medical School
Contact: melissa.dibacco@childrens.harvard.edu

Objective: Extracorporeal membrane oxygenation (ECMO) is used in newborns and children in the setting of life-threatening illness including

respiratory arrest, post-cardiac arrest care, and sepsis. These patients are high risk for cerebral injury and electrographic seizures (ES). We aim to describe the main clinical and electroencephalographic characteristics of neonates and children on ECMO, while focusing on the impact of seizures on outcome. Methods: Retrospective study of patients from birth to 21 years in the neonatal and pediatric intensive care unit (ICU) while requiring ECMO at Boston Children's Hospital from October 1, 2010 to September 30, 2017. cEEG was defined as > 3 hours on continuous monitoring. Subjects with seizures and brain injury prior to ECMO were excluded. Results: 436 patients (234 pediatric and 202 neonates) were included. The median age was 39 wks; 63% M and 2 yrs; 50% M for the neonatal and pediatric group respectively. The most common reason for ECMO was due to a cardiac etiology (268/436). 201 (109 pediatric and 92 neonates) had an cEEG while on ECMO. The percentage of patients on ECMO with cEEG increased from 12/51 in 2011 to 40/58 in 2017. Seizures were detected in 33/201 subjects with an EEG on ECMO, the majority detected in 2015 - 2017. Electrographic only seizures were seen in 22/33. Median seizure burden in the first 24 hours was 67 minutes. Mortality was high, 188/436 died. Mortality correlated to a higher seizure burden (p=0.042), and treatment for a clinical seizure prior to the initiation of EEG correlated to a lower seizure burden (p=0.047). Conclusions: Patients requiring ECMO are at extremely high risk for seizures that would go undetected without the use of cEEG. A lower seizure burden generally correlated with a more favorable outcome.
Number of hours per week: Generally student interns work 10 hours/week during the academic term. This is negotiable based on the arrangement made with each student. Requirements: Research is clinical research with some exposure to patients. No prior research experience in required.

Poster 81. High dimensional single cell characterization of cancer phenotypes and tumor microenvironment using t-CyCIF

Presenters: Rumana Rashid
Principle Investigator: Sandro Santagata
ssantagata@bics.bwh.harvard.edu
Laboratory for Systems Pharmacology at Harvard Medical School; Department of Pathology at Brigham and Women's Hospital
60 Fenwood Road, Room 8006-10 Boston, MA 02115
Contact: rrashid2@bwh.harvard.edu

Cyclic Immunofluorescence (CyCIF) facilitates multiplexed immunofluorescence imaging and single cell quantification of clinical samples. The method comprises multiple cycles of fluorescently tagged antibody incubation, imaging, and fluorophore inactivation. In the Lab for System Pharmacology (LSP), we are using CyCIF to characterize various cancer phenotypes including HSF1 foci formation, primary cilia expression, and nuclear rupture events. Furthermore, we are using this method to catalogue the immune profile and cell states of populations in tissue to gain a better understanding of the interaction between tumor cells and their microenvironment. We have used this method on multiple platforms including cultured cells, tissue sections, and tissue microarrays. By using CyCIF, we aim to get a more quantitative, granular, and spatially resolved understanding of the role of these phenotypes in oncogenesis to better inform cancer diagnostics and therapeutics. Number of hours per week: negotiable

Poster 82. Examining amino acid metabolism in leukemia: FLT3-ITD activates malic enzyme 2 and reprograms leukemia metabolism while inhibiting arginine metabolism in myelodysplastic syndrome reduces tumor growth.

Presenters: Una Choi, Eliane Grace
Principle Investigator: David Scadden, dscadden@mgh.harvard.edu
Harvard College, Sherman Fairchild 7 Divinity Ave Cambridge, MA
http://www.scaddenlab.com/
Contact: uchoi@college.harvard.edu

Currently, there is no cure for myelodysplastic syndromes (MDS) or acute myeloid leukemia (AML). Leukemia cells have aberrant glucose and amino acid metabolism; focusing on glutamine and arginine metabolism, then, could improve available treatments. 30% of AML cases involve mutations in the tyrosine kinase Fms-like tyrosine kinase 3 (FLT3). FLT3 mutations are associated with poor survival and most occur as internal tandem duplications (ITD), which constitutively activate FLT3 and promote glucose and glutamine consumption. As glucose and glutamine are the major carbon sources for proliferating cells, FLT3-ITD potentially modulates glucose and glutamine utilization in leukemia. Promisingly, malic enzymes (MEs) coordinate glutamine and glucose metabolism through catalyzing the oxidative decarboxylation of malate to pyruvate. While MEs maintain mitochondrial metabolism and are highly expressed in many tumors, their relationships with FLT3-ITD have not yet been characterized. We hypothesize that MEs are involved in FLT3-ITD-induced leukemia metabolism remodeling. Arginase has been

demonstrated to have an immunosuppressive effect on cancers. Arginase inhibitors reduce tumor growth by preventing the depletion of arginine in the tumor microenvironment. We will investigate arginase levels in hematopoietic stem and progenitor cells (HSPCs) in wildtype and MDS mouse models, and test the effect of an arginase inhibitor on normal and MDS HSPCs. We used flow cytometry to determine arginase protein levels in mouse HSPCs. Preliminary data suggest increased protein levels of ARG1 and ARG2 in MDS HSPCs. We will also determine arginase levels using ELISA. If differences can be confirmed, we will target arginase activity using an arginase inhibitor. If successful, our findings could lead to the first metabolic treatment for MDS. Number of hours per week: negotiable
Requirements: prior laboratory research experience required

Poster 83. BRAINcode and Beyond: Decoding Neuronal Genomes to Solve Neuropsychiatric disease

Presenters: Xianjun Dong
Principle Investigator: Clemens Scherzer
cscherzer@rics.bwh.harvard.edu
Center for Advanced Parkinson's Disease Research, Precision Neurology Program, Brigham and Women's Hospital, Harvard Medical School
60 Fenwood Rd, Boston, MA 02115, http://scherzerlaboratory.org/
Contact: xdong@rics.bwh.harvard.edu

Major questions about the awesome complexity and unparalleled processing capabilities of human brains remain mysterious. What specific code embedded in the three billion base-pairs of the human genome programs 86 billion neurons in our brain? What are the scripts running a dopamine or a pyramidal neuron? What are the glitches causing Parkinson's, Alzheimer's and other neuropsychiatric diseases that affect more than 50 million people around the world? To begin to answer these BIG questions, the BRAINcode project aims to provide a comprehensive map of actively transcribed genome elements, both protein-coding and non-coding, from specific neuron types, directly isolated from human brains of controls and across all stages of neuropathology. Phase 1.5 has sequenced total RNAs for >50,000 neurons from more than 200 human post-mortem brains. Most of what we known is based on the analysis of just the 1% of the human genome that encodes proteins. We found that cumulatively, 64.4% of the genome are massively transcribed into non-coding or "dark matter" RNAs in human brain. We discovered 71,022 novel transcribed noncoding

elements, many of which consistent with active enhancers, and validated both in vitro and in vivo. Surprisingly, genetic variants not for Parkinson's disease, but also schizophrenia, addiction, and sleep were enriched in these elements. Discoveries from this initial phase of BRAINcode were recently published in Nature Neuroscience (Dong et al., 2018), therapeutic clues explored in Science (Mittal et al., 2017), and predictive algorithms beta-tested in Lancet Neurology (Liu et al., 2017). We established a generally useful resource and easy-to-use web portal (http://humanbraincode.org) for evaluating targets for biomarkers and therapeutics in specific brain cell types. Do you want to do something amazing?
Number of hours per week: We recommend that during the academic term (Sept-May) Freshmen and Sophomores work 6-10 hours/week, Juniors and Seniors 15-20 hours/week. During the summer all students can work up to 40 hours per week. But it is negotiable, and depends on arrangement with each individual student.
Requirements: No prior research experience required. Having programming experience will be a plus.

Poster 84. Pro-Resolving mediators produced during coagulation activate human phagocytes intracellular single cell signaling in whole blood

Presenters: Stephania Libreros, Paul C Norris
Principle Investigator: Charle Serhan cserhan@bwh.harvard.edu
Brigham and Women's Hospital and Harvard Medical School
60 Fendwood Rd Hale Building of Transformative Medicine, Room 3012
Boston, MA 02115, http://serhanlab.bwh.harvard.edu
Contact: slibreros@bwh.harvard.edu

Phagocytes play a pivotal role during the acute inflammatory process by controlling host defense mechanisms, via temporal regulation of classical pro-inflammatory eicosanoids and the production specialized pro-resolving mediators (SPMs) that include resolvins, protecting and maresins. Coagulation is a host protective response to a barrier breach that can occur during infection. Here, we investigate the relationship between coagulation and the resolution of infectious inflammation using state of the art metabololipidomic-based profiling approach in human whole blood during coagulation. We identified temporal clusters of endogenously produced pro-thrombotic and pro-inflammatory lipid mediators (eicosanoids) as well as specialized pro-resolving mediators (SPMs) of resolvin E1, resolvin D1, resolvin D5, lipoxin B4, and maresin 1 each at bioactive [0.1-1.0 nM] concentrations. Using mass cytometry,

we present evidence that SPM evoke intracellular signaling pathways during host defense and coagulation at a single cell level using state-of-the-art time of flight mass cytometry (CyTOF) in human leukocytes. Nineteen phenotypic surface makers and eight phosphoepitopes intracellular proteins were analyzed using CyTOF in human whole blood samples incubated with or without SPM (RvE1, RvD1, RvD5, LXB4 and MaR1) cluster. High dimensional visualization t-Stochastic Neighbor Embedding (viSNE) unsupervised clustering analysis demonstrated that the SPM cluster produced in human whole blood targets leukocytes, directly activating signal transduction pathways at the single-cell level. This SPM cluster enhances containment and phagocytosis of Escherichia coli (E. coli) by neutrophils and monocytes. These studies identify a proresolving circuit, including endogenous molecular brakes and accelerators, which promoted host defense.
Number of hours per week: 6-10 or 15-20 hours/week. Hours and stipend are negotiable
Requirements: No prior research experience is required

Poster 85. Targeted Stem Cell therapies for Primary and Metastatic Brain Tumors
Presenters: Deepak Bhere
Principle Investigator: Khalid Shah, kshah@bwh.harvard.edu
Brigham and Women's Hospital /Harvard Medical School
60 Fenwood Rd. Boston, MA 02115, https://csti.bwh.harvard.edu
Contact: mmorris19@bwh.harvard.edu

The unique tumor homing capability of stem cells home has unveiled novel possibilities for their use in cancer therapy. Our research is based on targeting tumor cell specific molecular targets in an effort to eradicate both primary and metastatic tumors in the brain using therapeutically armed stem cells. We have engineered different cell surface receptor targeted adult stem cells to release (i) pro-apoptotic proteins to specifically induce apoptosis in tumor cells; (ii) anti-proliferative nanobodies (ENb) to inhibit tumor cell proliferation; (iii) immunomodulatory proteins to enhance T cell function; (iv) anti-angiogenic proteins to target blood vessels supplying the tumor; (v) oncolytic viruses to induce viral oncolysis; and demonstrated the therapeutic efficacy of these engineered stem cells both in vitro and in vivo. We employ various optical imaging techniques to track the fate of stem cells and tumor cells in real time in vivo. In an effort to develop clinically translatable therapeutics, we have developed and utilized immuno-deficient and -competent mouse tumor models that mimic

clinical settings of primary brain tumors and their secondary micro-invasive deposits in the brain. Our overall goal is to develop a multitude of targeted therapies that can be integrated into the standard of brain tumor therapy. Number of hours per week: During the academic term, we encourage students to commit to about 10-15hr/ week. This can be negotiated based on academic burden. Students are expected to work full time (40h) during the summer term. Requirements: Although prior research experience with molecular biology techniques is encouraged, a strong commitment and dedication to learn techniques is required. Students will receive training in various scientific areas including but not limited to experimental design, conduct, data interpretation and analysis, writing scientific reports and manuscripts.

Poster 86. Protecting Health of the Next Generation and Beyond: Studying environmental threats to the human genome using cell culture models of germline cells generated from the induced pluripotent stem cells

Presenters: Toshi Shioda, Keiko Shioda
Principle Investigator: Toshi Shioda, tshioda@mgh.harvard.edu
Harvard Medical School; Massachusetts General Hospital
Massachusetts General Hospital Center for Cancer Research,
https://www.massgeneral.org/cancerresearch/research/researchlab.aspx?id=1195

We are interested in genetics and epigenetics of human germline cells, which are the only lineage of cells that can covey genetic information to the next generation. DNA damage introduced into the genome of germline cells may result in creation of irreversible and permanently inherited mutations, which could cause familial diseases. For example, when young cancer patients receive DNA-damaging chemotherapy and survive, or victims of nuclear power plant accidents are exposed to substantial doses of ionizing irradiation, whether their offspring would possibly suffer from new heritable diseases or such a risk is practically negligible is an important, but still unanswered question. The epigenetic mechanisms of gene expression are subjected to a series of almost complete “resetting” processes during the early stages of germline development for refreshed start of a new generation of life, whereas potentially harmful retroviruses parasitizing in human genome must always be epigenetically suppressed. Aberrations in the epigenetic resetting in germline cells may cause disease predisposition in the next generation. Because access to developing germline cells in human embryos or fetuses is extremely challenging, we are generating cell

culture models of human primordial germ cells from normal or genetically engineered induced pluripotent stem cells (iPS cells) and investigating the molecular mechanisms of mutagenesis and the epigenetic resetting in human germline cells, with special emphasis on the relevance to creation of new familial diseases. We are seeking for highly motivated undergraduate lab members, who will be immediately involved in the ongoing research projects and receive hands-on trainings of human iPS cell culture, genome engineering, deep sequencing, bioinformatics, as well as basic molecular biology techniques.
Number of hours per week: Undergraduate researchers are requested to secure at least 10 hours per week for Freshmen/Sophomores, and 20 hours per week of work hours for Juniors and Seniors, for responsible involvement in research. Scheduling will be negotiable and relatively flexible. There is no specific limit of the project length.
Requirements: No prior research experience is required. Applications from new undergraduate researchers are encouraged.

Poster 87. Suicidality and emotional inhibitory control in dually diagnosed adolescents

Presenters: Eleanor Schuttenberg
Principle Investigator: Marisa Silveri, msilveri@mclean.harvard.edu
McLean Hospital Harvard Medical School
McLean Imaging Center 115 Mill St Belmont, MA 02478
http://nlamh.mclean.harvard.edu
Contact: eschuttenberg@partners.org

Inhibitory control, a critical aspect of self-regulation, is reduced in adolescents with substance use disorders and in individuals at an elevated risk for suicide attempts. Dual diagnosis refers to a substance use disorder co-occuring with at least one additional psychiatric diagnosis. The current study explores relationships between suicidality and inhibitory control in a sample of dually diagnosed adolescents, assessed upon their admission to the McLean Hospital Acute Residential Treatment program. All participants included in the current analyses reported some lifetime suicidal ideation. Participants (N=335, 196 female, age=16.9±1.2) performed an emotional Go-NoGo task to measure inhibitory control in the presence of distracting images that were emotionally negative, neutral, positive or scrambled. Results showed individuals who reported at least one lifetime suicide attempt (N=164) had significantly lower accuracy on inhibitory (NoGo) trials across all background conditions. In patients reporting suicidal ideation within the past month (N=201, 124 female, age=16.9±1.2), a significant

group x background interaction effect was observed (p=.012) with individuals reporting suicide attempts (N=69) within the past month showing significantly increased impulsive errors on negative background trials (p=.020) and neutral background trials (p=.025). These data demonstrate that inhibitory control measures may be helpful in identifying adolescents at highest risk for attempting suicide and that inhibitory control in negative and neutral emotional contexts is particularly associated with recent suicidal actions. Further studies are necessary to parse out relationships between suicidality, inhibitory control and emotion, in order to develop interventions that may help reduce suicidality in dually diagnosed adolescents. Number of hours per week: We require a minimum commitment of 8 hours per week. Requirements: We require that students receive academic credit for the work they do in our lab, and that they work a minimum of 8 hours per week.

Poster 88. The Laryngeal Force Sensor: Quantifying Force Metrics during Suspension Microlaryngoscopy Surgery

Presenters: Allen Feng, Phillip Song
Principle Investigator: Phillip Song, phillip_song@meei.harvard.edu
Massachusetts Eye and Ear / Harvard Medical School
243 Charles Street Boston, MA 02114
Contact: allen_feng@meei.harvard.edu

OBJECTIVES: To determine the relationship between force metrics and post-operative complications after suspension microlaryngoscopy (SML) through the development of a novel sensor. STUDY DESIGN: Prospective observational study. METHODS: The laryngeal force sensor (LFS) is a force sensor designed for microphonosurgery. Prospectively enrolled patients had force metrics recorded for each procedure. Pre and postoperative surveys were completed to assess for the development of tongue related symptoms (dysguesia, pain, paresthesia, and paresis) or dysphagia. Perioperative narcotic use was also recorded for each case and converted to IV morphine equivalents (ME). RESULTS: 82 patients have completed the study. In total, 36% developed post-operative tongue symptoms and 21% developed abnormal EAT-10 scores. The mean maximum force across all procedures was 164.7N (95%CI: 141.0–188.4 N, range: 48.5–402.6 N). Multiple logistic regression showed maximum force (OR1.15, 95%CI: 1.02–1.29, p=0.019) and female gender (30.1%, 95% CI: 22.7–37.5%, p<0.001) as significant predictors for development of tongue related symptoms. Maximum force was predictive of an abnormal post-operative EAT-10 score (OR1.03, 95%CI: 1.00–1.06, p=0.045). Univariate analysis demonstrated a positive correlation

between perioperative ME and total suspension time (p < 0.001) as well as total impulse (p = 0.007). On multiple linear regression, total suspension time was a significant predictive variable, with a marginal incremental increase of 0.273 mg of ME per minute of suspension time (0.273 mg/min, 95% CI: 0.040 - 0.507 mg/min, p = 0.022).
CONCLUSIONS: The LFS is predictive of both postoperative complications and perioperative narcotic requirements. Future studies include multi-institutional trials, further device development, and assessment of long-term outcomes.
Number of hours per week: Hours per week are negotiable and can be tailored to each individual student.
Requirements: No prior research experience required, however experience can be tailored to each student. Preferrence will be given to applicants with experience or working knowledge of statistics (ex. STATA), coding (ex. C++, Java, Ptyhon, etc.), and running clinical studies.

Poster 89. Using a New Model System to Study the Mechanisms and Evolution of Regeneration

Presenters: Julian Kimura, Alyson Ramirez
Principle Investigator: Mansi Srivastava, mansi@oeb.harvard.edu
Department of Organismic and Evolutionary Biology
52 Oxford Street, Room 202, Northwest Laboratories,
www.srivastavalab.org
Contact: rhulett@g.harvard.edu

Wound repair and regeneration are fundamental features of animal biology. In humans and other vertebrates, virtually all wounds can be healed yet only some will result in subsequent regeneration of tissues lost to injury (e.g., liver in humans, tail fin in fish). In contrast, many other animal species can regenerate extensively and have the capacity to replace all tissues of the adult animal (e.g., planarians, cnidarians). Underlying these differential regenerative capacities are different stem cell systems; highly regenerative species often harbor pluripotent adult stem cells that can differentiate into virtually any cell type, whereas vertebrates have lineage-restricted adult stem cells. We seek to identify the molecular mechanisms that control specification, maintenance, and differentiation of pluripotent stem cells in vivo. We focus our studies on the acoel worm *Hofstenia miamia*, a highly regenerative member of the sister lineage to all other bilaterians, that has a large population of pluripotent stem cells called neoblasts. Hofstenia offers many advantages as a new model system for mechanistic studies of

regeneration, including accessible and manipulable embryos. We obtained a detailed atlas of developmental stages in *Hofstenia*, which is enabling us to identify the embryonic origins of neoblasts. Next, we developed methods for CRISPR/Cas9-mediate genome editing and for driving expression of fluorescent proteins under specific promoters in Hofstenia, which we will use to study the dynamics of the neoblast population during regeneration. Further, we have also applied single-cell RNA sequencing methods to identify regulators of neoblasts in regenerating animals. We also utilized Hofstenia's relationship to other animals to study the evolution of mechanisms of stem cells and regeneration. Number of hours per week: 12-15 hours/week Requirements: No prior research experience required.

Poster 90. Therapeutic and Diagnostic Strategies for Pediatric and Adult Brain Cancer

Presenters: Jian Teng, Markus Schweiger
Principle Investigator: Bakhos Tannous
bakhos_tannous@hms.harvard.edu
Department of Neurology, Neuro-Oncology Division, Massachusetts General Hospital Neuroscience Program, Harvard Medical School Neuroscience Center at Massachusetts General Hospital Massachusetts General Hospital – East Building 149, 13th Street Charlestown, MA
https://www.massgeneral.org/neurology/research/researchlab.aspx?id=1483
Contact: JTENG2@mgh.harvard.edu

Despite extensive treatment, long-term survival for pediatric and adult primary gliomas and brain metastases remains uncommon. Our work focuses on developing novel diagnostics and therapeutic strategies against tumors in the brain using three different strategies: (1) High Throughput Screening: We have developed a high-throughput drug screening pipeline that allows for the evaluation of thousands of different compounds against pediatric and adult glioblastomas and are testing a handful of promising drugs in patient-derived pre-clinical models. Further, by repurposing old drugs, we could show that Hydroxyurea, used to treat sickle cell disease, improves survival of mice with glioblastomas and we are currently testing this therapy in a clinical trial. (2) Gene and Cell Therapy: We develop novel gene/cell therapeutic approaches using (a) different viral vectors or stem cells to delivery anti-cancer therapeutics and (b) test the novel role of olfactory cells for anti-glioma therapy through intra-nasal injection, their natural route to CNS. (3) Diagnostics: Our work elucidates the role of extracellular vesicles and tumor-educated

platelets in brain tumor metastasis/progression and therapeutic resistance. In addition, we utilize the power of platelets to develop a novel diagnostic platform and to deliver therapeutic agents against brain tumors. Number of hours per week: negotiable, depending on arrangement with each individual student.
Requirements: no prior research experience required.

Poster 91. Non-coding RNAs, RNPs & Translation in Cancer
Presenters: Syed Irfan Ahmad Bukhari, Samuel Spencer Truesdell
Principle Investigator: Shobha Vasudevan
vasudevan.shobha@mgh.harvard.edu
MGH Cancer Center, Harvard Medical School, Harvard Stem Cell Institute, Center for Regenerative Medicine
MGH, Main Campus, Simches Research Bldg, 185 Cambridge St, CPZN 4100/4202, Boston, MA
http://dms.hms.harvard.edu/BBS/fac/Vasudevan.php
Contact: shobhavas@gmail.com

Quiescent (G0) cancer cells are dormant, reversibly-arrested cells, including stem cells, which resist clinical therapy that eliminates proliferating cancer cells. Upon chemotherapy removal, G0 cells sense the loss of their proliferating neighbors and restart cell division, restoring the cancer as recurrence. G0 shows a switch to a distinct gene expression program where RNA regulation enables persistence of this critical state. mRNA control elements and regulatory RNAs such as non-coding RNAs and microRNAs, interact with RNA binding proteins to form RNA-protein complexes (RNPs) and direct expression of clinically relevant genes; their deregulation leads to a range of clinical effects such as tumor resistance, immune and developmental disorders. The primary goal of our research program is to investigate non-coding RNA- and RNA binding protein-controlled expression of critical genes in tumors, which lead to resistance and tumor expansion. A second focus is to characterize the mechanisms of expression or translation of critical genes in G0 states in cancers, stem cells and germ cells. A third aim is to develop therapeutic approaches to modulate RNA-controlled expression in tumor resistance. These investigations will provide insights and novel therapeutics on non-coding RNAs in tumor resistance.
Vasudevan Lab Ph: 617-643-3143
vasudevan.shobha@mgh.harvard.edu
http://dms.hms.harvard.edu/BBS/fac/Vasudevan.php
Number of hours per week: Negotiable especially around classes, exams and other career goals and priorities.

Requirements: Appreciate prior research experience but its not necessary. We do insist on persistent, diligent students who want to keep learning.

Poster 92. Protein Motion and Configurations in a Form-Fitting Nanopore: Avidin in ClyA
Presenters: Rebecca Frank, Louis Colson
Principle Investigator: Lene Vestergaard Hau, hau@g.harvard.edu
SEAS - Applied Physics
19A Oxford St. Cambridge, MA 02138,
https://www.seas.harvard.edu/haulab/index.htm
Contact: rebecca_frank@g.harvard.edu

We probe the molecular dynamics and states of an avidin protein as it is captured and trapped in a voltage-biased cytolysin A nanopore using time-resolved single-molecule electrical conductance signals. The data for very large numbers of single-molecule events are analyzed and presented by a new method that provides clear visual insight into the molecular scale processes. Avidin in cytolysin A has surprisingly rich conductance spectra that reveal transient and more permanently trapped protein configurations in the pore and how they evolve into one another. We identify a long-lasting, stable, and low-noise configuration of avidin in the nanopore into which avidin can be reliably trapped and released. This may prove useful for single-molecule studies of other proteins that can be biotinylated and then transported by avidin to the pore via their coupling to avidin with biotin-avidin linking. We demonstrate the sensitivity of this system with detection of biotin attached to avidin captured by the pore.
Number of hours per week: To be arranged on a case by case basis
Requirements: N/A

Poster 93. High-throughput computational microscopy for quantitative biology
Presenters: Dushan Wadduwage
Principle Investigator: Dushan Wadduwage
wadduwage@fas.harvard.edu
Center for Advanced Imaging, J. Harvard Distinguished Science Fellows
Wadduwage Lab, Center for Advanced Imaging Northwest Building, 52 Oxford St., Suite 147, Room 146.10,

Today Biology has become an increasingly quantitative science. Being the key tool in bio-imaging, microscopy too is routinely used in

quantitative biological studies, generating large-scale image datasets. However, this comes at a cost of longer image acquisition time. In this poster we present our approach to address this problem by seamlessly combining optics and computational tools to cut-down image acquisition time. We first present a general computational optics framework for biological microscopy. Secondly, we present two examples of computational microscopy systems: a high-throughput hyper-spectral imaging system, and a high-throughput two-photon imaging system. The former is a powerful tool for multiplexed biological fluorescence imaging and Raman imaging. The latter has applications in neuro-biology to image brain activity in live animals. Both these systems are an order to two orders of magnitude faster than the current state of the art.
Number of hours per week: Negotiable.
Requirements: No prior research experience is required.

Poster 94. A Conserved Role for the N-Glycosylation Pathway in Sleep and Seizures

Presenters: Brittany Leger, James Walker
Principle Investigator: James Walker, jwalker@helix.mgh.harvard.edu
Center for Genomic Medicine, Massachusetts General Hospital, HMS
Center for Genomic Medicine Massachusetts General Hospital Simches Research Building, Room 5600 185 Cambridge Street Boston, MA
https://www.massgeneral.org/neurology/research/researchlab.aspx?id=1794
Contact: BSLEGER@mgh.harvard.edu

Protein N-glycosylation is a post-translational modification important for the folding, stability and secretion of proteins. Assembly of the precursor glycan is carried out by a set of ALG enzymes. Mutations that disrupt the function of these ALG enzymes cause a family of rare metabolic disorders called congenital disorders of glycosylation. GWAS of human sleep disturbance revealed variants near two paralogous ALG enzyme coding genes, ALG10 and ALG10B, affecting multiple sleep and chronotype traits. Further, a patient with an ultra-rare quadruple KO mutation for ALG10 and ALG10B showed epilepsy, among other phenotypes. To determine the directionality of both the seizure and the sleep effects, the precise sleep/chronotype parameter that is altered, and the causal gene in these haplotypes, we used Drosophila as a model system. While Drosophila has only one Alg10 gene, no phenotype has yet been reported. Neuron-specific Alg10 knockdown using RNAi lines results in sleep defects and seizures. To verify a role for Alg10 in fly seizures and sleep, we have used imprecise excision of a P-element

insertion and CRISPR/Cas9 gene editing to generate a series of Alg10 mutants, which are being behaviorally characterized. Additionally, transgenic flies expressing human ALG10 and ALG10B are being used to rescue the fly RNAi phenotypes. In summary, a novel gene (ALG10) and pathway (N-glycosylation) plays a role in human and Drosophila sleep and seizures, that has been conserved across almost a billion years of evolution. Future mechanistic studies using chemical biological tools will identify target protein(s) whose glycosylation is disrupted in Alg10 mutant flies leading to neurological deficits and will be investigated for a role in human sleep using pre-existing cohort-scale data.
Number of hours per week: Hours are negotiable and flexible, depending on the experiments being conducted at the time. Minimum expectations are 2-3 hours, 2-3 days a week. Length of project will be a minimum of 6 months but can be extended. Requirements: Some laboratory experience is preferred (molecular/protein biochemistry), but full training will be given. Must be self-motivated with attention to detail and excellent verbal and written communication skills. Duties include maintenance of fly stocks and genetic crosses, cell culture, microscopy, CRISPR gene-editing, molecular biology and protein techniques including: DNA/RNA isolation, PCR and sequencing, western blotting.

Poster 95. Harnessing NV Quantum defects in Diamond for applications in physics, biology, and chemistry

Presenters: Erik Bauch, Connor Hart
Principle Investigator: Ronald Walsworth, rwalsworth@cfa.harvard.edu
Harvard Physics, Harvard Center for Astrophysics, Harvard Center for Brain Sciences (CBS)
60 Garden Street, 02138 Cambridge, US, http://walsworthlab.org
Contact: bauch@g.harvard.edu

Physicists find great scientific value in diamonds with tiny imperfections known as NV centers. Diamond consists of a crystalline array of carbon atoms. When one carbon atom is replaced with a nitrogen atom, and there is a missing atom or “vacancy” nearby, this forms what is known as a nitrogen-vacancy (NV) center. The atomically small size of an NV center enables physicists to probe electromagnetic fields on a miniscule scale currently inaccessible with other systems. One application is the sensing of magnetic fields: NV centers have spin magnetic moments, which rotate in an external magnetic field, like a compass needle aligning along the Earth’s magnetic field. When excited by a green laser, NV centers fluoresce red light, and the brightness of this light depends on the direction the NV spins are pointing. Physicists can then measure a

magnetic field's direction and strength by monitoring changes in the red light emitted by NV centers. Our group consists of more than 20 Ph.D. students, undergraduates, and postdoctoral fellows, all with a focus on developing cutting-edge measurement tools and applying them to important problems in both the Physical and Life Sciences. Current areas of research in our group include among others: nanoscale magnetometry and spin physics; imaging of biomagnetism, solid-state physics, and the development of novel magnetic-resonance-imaging (MRI) tools, with applications to medical imaging. Check out our website for more information and select publications. Number of hours per week: negotiable. Requirements: some basic EE experience would be nice; good physics knowledge (Quantum 144 if possible).

Poster 96. The molecular mechanisms controlling osteocyte differentiation and function

Presenters: Tadatoshi Sato
Principle Investigator: Marc Wein, mnwein@mgh.harvard.edu
Harvard Medical School, Massachusetts General Hospital, Endocrine Unit
50 Blossom Street, Thier Building / Boston MA, 02114
https://scholar.harvard.edu/wein
Contact: satotadatoshi@gmail.com

The Wein laboratory studies molecular mechanisms controlling osteocyte differentiation and function. Our research focuses on transcriptional control of sclerostin, an osteocyte-specific secreted protein that happens to be a drug target for osteoporosis. We are interested in understanding how osteocytes turn on sclerostin, and how signals that boost bone formation (parathyroid hormone, mechanical loading) regulate its expression. **Parathyroid hormone signaling** We recently found that PTH signaling in osteocytes works in large part by turning off the kinase SIK2 (Wein et al. Nat Commun. 2016). Small molecule SIK2 inhibitors mimic PTH action, both in cultured osteocytes and in vivo. Now we are further investigating the PTH/SIK signaling axis in osteocytes, and pursuing small molecule SIK inhibitors as agents to treat osteoporosis. In addition, we are exploring whether salt inducible kinases participate in PTH action in other target organs. Mechanisms of mechanotransduction in osteocytes It is known that osteocytes respond to mechanical cues, but exactly how this occurs is quite mysterious. We are using genomic and proteomic approaches to better understand the intracellular signaling pathways through which osteocytes sense and respond to mechanical forces. Now we are testing how HDAC4/5

participate in osteocyte mechanotransduction to regulate SOST inhibition and bone formation. **Osteocytogenesis** Osteoblasts synthesize bone matrix and differentiate into osteocytes. The molecular pathways responsible for this fascinating differentiation event are unknown. We are using unbiased approaches to try to better understand the process of osteocytogenesis, with the goal of identifying novel transcriptional regulators and signaling pathways that allow for this dramatic cellular transformation.
Number of hours per week: Freshmen and Sophomores work 6-10 hours/week, Juniors and Seniors 15-20 hours/week. During the summer all students can work up to 40 hours per week. It is negotiable.
Requirements: These skills are preferable but not required; Cell culture, qPCR, RNA isolation, protein extraction and western blotting.

Poster 97. Information acquisition as a biomarker for vision impairment

Presenters: Francisco Costela
Principle Investigator: Russell Woods, russell_woods@meei.harvard.edu
Schepens Eye Research Institute / Harvard Medical School
20 Staniford St, Boston, 02114,
Contact: francisco_costela@meei.harvard.edu

Introduction: People with vision impairments report difficulties watching video (e.g. television and movies). To our knowledge, no studies have objectively assessed this difficulty. Recently, we developed an objective measure of the ability to follow the story in video, the information acquisition (IA) metric. Here, we test whether IA can measure the impact of vision impairment. Methods: Participants with central visual loss (CVL, reduced visual acuity, N=21), with hemianopia (loss of vision on one side, N=24) or normal vision (NV, N=60) watched 30-second Hollywood video clips, then described the clip content. An objective score was derived using natural-language processing to compare each new response to a control database of descriptions of the same clip. To test whether the impact of CVL was simply due to reduced resolution, NV participants (N=15) watched the same clips with defocus blur. Results: Compared to the NV group, IA was worse for the CVL (z=-4.63, p<0.001) and hemianopia (z=5.78, p<0.001) groups. In our sample, IA was not higher for the hemianopia than the CVL group (-0.83, p=0.409), despite better visual acuity. The NV-blur group had better IA than the CVL group (z=3.95, p<0.001). Conclusion: The IA method was able to measure the difficulty following the story experienced by people with CVL and hemianopia. Also, IA showed that defocus blur

failed to recreate the CVL experience. These results confirm that IA can be used to measure the impact of vision impairment on the video-viewing task, and is likely to be useful for measurement of the effect of vision rehabilitation. Number of hours per week: It is negotiable
Requirements: No prior research experience is required

Poster 98. The HOPE Pilot Study: Harnessing Patient-Reported Outcomes and Biometric Data to Enhance Cancer Care
Presenters: Elizabeth Schrier, Embree Thompson
Principle Investigator: Alexi Wright, alexi_wright@dfci.harvard.edu
Harvard Medical School, Dana-Farber Cancer Institute
Dana-Farber Cancer Institute, Dana 1110, 44 Binney Street, Boston MA 02215,

Purpose: Integrating patient-reported outcomes (PROs) into clinical practice is an increasingly promising strategy for improving patients' symptoms, communication, and clinical outcomes. The objective of this study was to assess the feasibility, acceptability, and perceived effectiveness of a mobile health intervention designed to collect PROs and activity data as a measure of health status. Patients and Methods: Pilot intervention with 10 patients with gynecologic cancers receiving palliative chemotherapy. The HOPE (Helping Our Patients Excel) study used wearable accelerometers to assess physical activity and the Beiwe research platform to collect PROs, risk stratify patient responses, provide tailored symptom management, and notify patients and clinicians of high-risk symptoms. Feasibility and acceptability were assessed through enrollment and adherence rates; perceived effectiveness was evaluated by patients and oncologists at study completion. Results: The approach-to-consent rate was 100% and participants were 90% and 70% adherent, respectively, to the wearable accelerometers and smartphone surveys. Participants' mean daily step count was 4,040 (SD=2,270) and increased from week 1 (mean=3,520, SD=1578) to week 3 (mean=4,136, SD=1,578). Active monitoring of participants' heart rates, daily steps, and PROs throughout the study identified anomalies in participants' behavior patterns that suggested poor health for 20% (2 patients). Patients and clinicians indicated that the intervention improved physical activity, communication, and symptom management. Conclusion and Relevance: A mobile health intervention that collects PROs and activity data as a measure of health status is feasible, acceptable, and was perceived to be effective in improving symptom management patients with advanced gynecologic cancers. A larger,
Number of hours per week: 15-20 hours but negotiable

Requirements: Ideal candidates will have an interest in qualitative research analysis or computer coding experience

Poster 99. Clinical Computational Neuroimaging Laboratory at the Athinoula A. Martinos Center for Biomedicla Imaging

Presenters: Brandon Hancock
Principle Investigator: Ona Wu, ona@nmr.mgh.harvard.edu
Massachusetts General Hospital
Athinoula A. Martinos Center for Biomedical Imaging 149 13th Street CNY 2301 Charlestown, MA 02129, http://www.martinos.org/lab/ccni
Contact: ona@nmr.mgh.harvard.edu

The Clinical Computational Neuroimaging Group is a team of interdisciplinary individuals interested in both data science and clinical research. Our research activities focus on developing methods to improve the diagnosis, prognosis and management of patients with brain injury as a result of stroke, cardiac arrest or trauma. We use state-of-the-art MRI acquistion and analysis techniques and combine imaging with clinical data via machine learning algorithms to create quantitative biomarkers that can be used to monitor disease status relative to "normal" tissue or evaluate progression or recovery. We will present some of our research areas. We use imaging to triage patients with unwitnessed acute ischemic stroke for thrombolytic therapy, offering treatment options to patients for whom currently none exist. We also develop techniques to predict tissue and clinical outcome after acute ischemic stroke and to evaluate therapeutic efficacy of novel stroke treatments. For comatose cardiac arrest patients, we apply imaging to predict long-term neurological outcome by investigating changes in post-arrest structural and functional brain connectivity. We also use algorithms to combine structural and functional MRI to evaluate the sequelae of post-concussive mild traumatic brain injury.
Number of hours per week: Negotiable
Requirements: Experience with Linux operating systems preferred.

Poster 100. Mapping the wiring of the human brain with diffusion MRI

Presenters: Nivedita Ravi, Chiara Maffei
Principle Investigator: Anastasia Yendiki, ayendiki@mgh.harvard.edu
Martinos Center for Biomedical Imaging, Massachusetts General Hospital and Harvard Medical School
Martinos Center for Biomedical Imaging, 149 Thirteenth Street, Rm 2301 Charlestown, MA 02129, https://scholar.harvard.edu/a-y

Contact: cmaffei@mgh.harvard.edu

Our group works on mapping the connectional anatomy of the human brain. We use very high-resolution MRI and optical imaging techniques that are only feasible ex vivo to create accurate models of brain pathways from post mortem human brains. We then use these models to train algorithms that can reconstruct the same connections from the much lower resolution MRI brain scans that can be performed on living subjects. Our goal is to develop computational tools that allow clinicians and researchers to study how the brain is wired in healthy subjects as well as patients with psychiatric and neurological disorders. As an example of this work, we show our efforts to map the superior longitudinal fasciculus (SLF), a major antero-posterior brain pathway. The SLF is composed of three subcomponents, which have been described using invasive, autoradiographic techniques in monkey brains. There have been efforts to reconstruct the SLF in humans based on non-invasive diffusion MRI scans, but studies have yielded conflicting results, and there are still controversies in the literature. Here we use data from the Human Connectome Project to illuminate these controversies and to suggest optimal macroanatomical landmarks for the virtual dissection of this pathway, which is implicated in many disorders.
Number of hours per week: Negotiable depending on project
Requirements: Some of our projects require prior experience in computer programming. However there are also projects available that do not require prior experience.

Poster 101. Hijacking Host Kinases for HIV-1 Phosphorylation
Presenters: Fatema Chowdhury
Principle Investigator: Xu Yu, xyu@partners.org
Ragon Institute of MGH, MIT and Harvard
400 Technology Square Cambridge, MA 02139
http://www.ragoninstitute.org/portfolio-item/yu-lab/
Contact: fchowdhury@mgh.harvard.edu

As a small virus with restricted genome size, HIV-1 critically depends on host machinery to support its life cycle. Upon infecting CD4+ cells, HIV-1 integrates itself into the host genome and replicates itself, all the while interacting with host cellular components. One such interaction involves protein phosphorylation cascades which is yet to be characterized in the context of HIV-1 proteins. Phosphorylation is known to regulate protein folding, localization, activity, half-life, and interaction with other molecules. Host kinase-mediated phosphorylation thus may regulate

mechanisms indispensable to viral replication and virulence. In collaboration with the Broad Institute, we have used highly-sensitive mass spectrometry-based phosphoprotein profiling approaches for proteome-wide mapping of HIV-1 phosphorylated sites in infected CD4+ T cells as well as cell-free virus. We have detected over 30 unique phosphorylated sites on HIV-1, few of which also serve as targets for immune response. This is the first study to comprehensively map global landscape of phosphorylated residues on HIV-1 proteins. We are continuing to characterize the functional impacts of these viral protein modifications such as effects on viral fitness, which in turn could be targeted by pharmacologic or immunologic interventions.
Number of hours per week: Negotiable and also depends on arrangement with each individual student.
Requirements: No prior laboratory experience is required other than willingness to learn.

Poster 102. Decomposing spatially dependent and cell type specific contributions to cellular heterogeneity
Presenters: Qian Zhu
Principle Investigator: Guo-cheng Yuan, gcyuan@jimmy.harvard.edu
Dana-Farber Cancer Institute Harvard School of Public Health
LC1060 Dana-Farber Cancer Institute 450 Brookline Avenue,
http://bcb.dfci.harvard.edu/~gcyuan/
Contact: zqian@jimmy.harvard.edu

Both the intrinsic regulatory network and spatial environment are contributors of cellular identity and result in cell state variations. However, their individual contributions remain poorly understood. Here we present a systematic approach to integrate both sequencing- and imaging-based single-cell transcriptomic profiles, thereby combining whole-transcriptomic and spatial information from these assays. We applied this approach to dissect the cell-type and spatial domain associated heterogeneity within the mouse visual cortex region. Our analysis identified distinct spatially associated signatures within glutamatergic and astrocyte cell compartments, indicating strong interactions between cells and their surrounding environment. Using these signatures as a guide to analyze single cell RNAseq data, we identified previously unknown, but spatially associated subpopulations. As such, our integrated approach provides a powerful tool for dissecting the roles of intrinsic regulatory networks and spatial environment in the maintenance of cellular states.

Poster 103. Tissue model biofabrication with microfluidic chips and bioprinting

Presenters: Nan Jiang, Guoliang Ying
Principle Investigator: Yu Shrike Zhang
yszhang@research.bwh.harvard.edu
SEAS and Harvard Medical School
Mckay building, 5th floor, Cambridge MA 02138 65 Landsdowne street, Cambridge MA 02139, https://www.shrikezhang.com/;
https://weitzlab.seas.harvard.edu/
Contact: nanjiang1@seas.harvard.edu

Regenerative medicine has been gained tremendous progresses over the past few decades due to its ability to fabricate functional simulated tissue constructs. Existing methods including scaffolding and microengineering are restricted by precisely producing biomimetic tissue constructs. Biofabrication techniques, including microfluidic based organs on chips and three-dimensional (3D) bioprinting, provide a connection to bridge artificially engineered tissue constructs and native tissues. Moreover, biofabrication technique offers unprecedented versatile platforms to co-deliver living cells and biomaterials with precise control over their constituents and spatial distributions, for personalized reconstruction of the fine structure, architecture, and functionality of target human tissues or organs. Our on-going projects are focused on both of microfluidic-based organ on chips and 3D bioprinting for biomimetic constructs of several representative tissues and organs, such as blood vessel, heart, liver, and cartilage. We are aiming at achieving the goal of accelerating drug discovery and personalized medicine.
Number of hours per week: 15 hours per week
Requirements: no

Poster 104. Anatomically-guided streamline clustering in newborns using AnatomiCuts

Presenters: Lilla Zollei, Viviana Siless
Principle Investigator: Lilla Zollei, lzollei@nmr.mgh.harvard.edu
Laboratory of Computational Neuroimaging, A A Martinos Center, Massachusetts General Hospital
149 13th St Charlestown, MA 02129, https://www.martinos.org/lab/lcn
Contact: lzollei@nmr.mgh.harvard.edu

White matter analysis is a challenging task in the infant brain due to ongoing postnatal myelination, potential motion artifacts and lower voxel resolution resulting from shorter acquisitions that may all lead to false

positives in tractography outputs. Moreover, optimizing spatial registration algorithms for this age group is also non-trivial. We apply AnatomiCuts, a tractography clustering algorithm based on anatomical similarity and evaluate its performance using Infant Freesurfer parcellations. Relying on anatomical similarity enables our analysis to be completed without computing spatial alignment into a common space. Our data set of interest is the 40 newborns from the first data release of The Developing Human Connectome project (www.developingconnectome.org) (dHCP).
Number of hours per week: This is negotiable, however, preferred time commitment during the academic term is 8 hrs / week and during the summer 40 hrs / week.
Requirements: Familiarity with Unix / Linux and scripting (csh/bash/python) is a plus. Knowledge of neuroanatomy also provides a great advantage.

Poster 105. Biology of Hematopoiesis and Cancer
Presenters: Meredith Stanhope, Marlies Rossmann
Principle Investigator: Leonard Zon leonard.zon@enders.tch.harvard.edu
Stem Cell Program and Division of Hematology/Oncology at Boston Children's Hospital and Dana Farber Cancer Institute, Howard Hughes Medical Institute, Harvard Medical School, Harvard Stem Cell Institute, Harvard Stem Cell and Regenerative Biology
Sherman Fairchild Biochemistry Building Ground Floor 7 Divinity Ave Cambridge, MA 02138,
https://proxy.qualtrics.com/proxy/?url=https%3A%2F%2Fzon.tchlab.org&token=MOffTYIkVUDl2fdBoRayBnrdy9DYaGfItc9Wi%2FGxV%2BE%3D
Contact: meredith_stanhope@fas.harvard.edu

The Zon laboratory focuses on the developmental biology of hematopoiesis and cancer in zebrafish and mouse systems, as well as human cell lines. We have collected well over 30 mutants affecting the hematopoietic system. Some of the mutants represent excellent animal models of human disease. Recently, we identified several chromatin factors with essential roles at various stages of hematopoiesis. We have developed suppressor screening genetic methods and found that transcriptional elongation regulates blood cell fate. We also have performed chemical genetic screens to identify components implicated in blood development and have found that prostaglandins increase the number of blood stem cells. This has led to a clinical trial to improve engraftment for patients receiving cord blood transplants; phase I trials were just successfully completed. The laboratory has also

developed zebrafish models of cancer. Using transgenics, we have generated a melanoma model in the zebrafish system. Transgenic fish get nevi, and when combined with a p53 mutant fish develop melanomas. We recently found a histone methyltransferase that can accelerate melanoma, and discovered a small molecule that blocks transcription elongation and suppresses melanoma growth.
Number of hours per week: Negotiable: depends on arrangement
Requirements: Prior lab experience is recommended but not required.

Informational Tables

Table 1. Life Sciences Concentration Advising & Undergraduate Research Advising

Life Science Concentration Advising
Biomedical Engineering: Lindsey Moyer, lmoyer@seas.harvard.edu, 206C Pierce Hall
Chemical and Physical Biology: Dominic Mao, dominicmao@fas.harvard.edu, Sherman Fairchild Room 195
Chemistry: Gregory Tucci, tucci@fas.harvard.edu, Science Center, Room 114
Cognitive Neuroscience and Evolutionary Psychology, a track in the **Psychology concentration:** Katherine Powers, kpowers@fas.harvard.edu, William James Hall, Room 218
Human Developmental and Regenerative Biology: Bill Anderson, wanders@fas.harvard.edu, Bauer Laboratory Room 204
Human Evolutionary Biology: Carole Hooven, hooven@fas.harvard.edu**,** Peabody 52F; Bridget Alex, balex@fas.harvard.edu, Peabody 53B
Integrative Biology: Andrew Berry, berry@oeb.harvard.edu, BioLabs Room 1082B
Molecular and Cellular Biology: Dominic Mao, dominicmao@fas.harvard.edu, Sherman Fairchild Room 195
Neurobiology: Ryan Draft, draft@fas.harvard.edu, Biolabs Room 1082A; Laura Magnotti, magnotti@fas.harvard.edu, BioLabs Room 1082D

Undergraduate Science Research Advising
Many Harvard undergraduates participate in science research at one of Harvard's campuses. If you are interested in research, the Undergraduate Science Research Advisor, Anna Babakhanyan, can help you navigate the process of finding a research group. She can help you: define your research interests, navigate research group websites, create and edit a science resume and cover letter, identify and contact research groups, interviews, apply for fellowships and funding, integrate effectively into your research group and make the most of your research experiences.

Contact: Dr. Anna Babakhanyan, ababakhanyan@fas.harvard.edu
http://lifesciences.fas.harvard.edu/research

Table 2. Mentoring Undergraduates: a Workshop Certificate Series for Scientist-Mentors

Margaret A. Lynch, PhD, Associate Director of Science Education
Anna Babakhanyan, PhD, MBT, Undergraduate Science Research Advisor
Science Education, Faculty of Arts & Sciences Division of Science
https://lifesciences.fas.harvard.edu/mentoring-workshops

Since 2008, our group has offered a certificate series of mentoring workshops for scientist-researchers who mentor or plan to mentor undergraduates conducting science research. Our highly interactive, case study-based workshops are designed to provide training, a forum for discussion, and support for these researchers. The workshop series is comprised of two workshop intensives followed by the series capstone, which is an interactive panel session with experienced undergraduate science researchers.

Participants can attend any or all of the workshops. Participants who complete the entire 11-hour workshop series receive a certificate of completion and distinction at an award ceremony. In workshop evaluations, participants stated that they increased their knowledge of effective undergraduate mentoring strategies.

2018-2019 Workshop Dates

Part 1: Undergraduate Research Fellowships & Writing Recommendation Letters / Developing Research Projects with Undergraduates/Diversity & Inclusion

Participants sign up for one of the following sessions:

Harvard Cambridge campus:
December 4, 2018 or January 9, 2019
9:30 am - 2:00 pm (light lunch provided)
Northwest Laboratories, 52 Oxford Street, Room 353

Harvard Medical School campus
January 16, 2019
10:00 am – 2:30 pm (light lunch provided)
tentative location: TMEC 328, 260 Longwood Avenue, Boston, MA 02115

Part 2: Mentoring Undergraduates: Handling Challenges/Celebrating Successes/Diversity & Inclusion/Supporting Student Science Writing

Participants sign up for one of the following sessions:

Harvard Cambridge campus:
December 11, 2018 or February 27, 2019
9:30 am - 2:00 pm (light lunch provided)
Northwest Laboratories, 52 Oxford Street, Room 353

Harvard Medical School campus
January 16, 2019
10:00 am – 2:30 pm (light lunch provided)
Waterhouse Room, Gordon Hall, HMS

Part 3: Undergraduate Researcher Panel: TBD

Table 3. Harvard Stem Cell Institute Internship Program
10-week program of laboratory experience in stem cell science, complemented with mandatory weekly stem cell companion course and closing symposium featuring oral and poster presentations by summer interns. Program accepts Harvard undergraduates as well as students from colleges and universities across the globe.
Contact: Maureen Herrmann, maureen_herrmann@harvard.edu, www.hsci.harvard.edu/internship
7 Divinity Avenue Bauer Building, Room G02A Cambridge, MA 02138

Table 4. Office of Undergraduate Research and Fellowships
The Office for Undergraduate Research & Fellowships (URAF) helps Harvard College students navigate the broad array of institutional, domestic, and international research opportunities that are available to them. The office further aims to develop connections among stakeholders in the academic research landscape (schools, academic departments and affiliated research enterprises, the housing communities, and student organizations).

URAF holds walk-in advising hours Monday through Friday from 2:00pm to 4:00pm on the second floor of 77 Dunster Street, and at other times by appointment (telephone: 617-495-5095). Information about conducting research as an undergraduate—including how to get started, your responsibilities as a researcher, and links to research opportunities and

funding at Harvard and beyond—may be found at the URAF website: https://uraf.harvard.edu/.

URAF offers the Program for Research in Science and Engineering (PRISE), the Harvard-Amgen Scholars Program, the Herchel Smith-Harvard Undergraduate Science Research Program, and the Harvard College Research Program (HCRP) for students interested in research in the life sciences. Below you can read more about each of these programs.

PRISE is a ten-week summer program that aims to build community and stimulate creativity among Harvard undergraduate researchers in the life, physical/natural, engineering, and applied sciences. To participate in PRISE, you must find a research position on your own, and apply to PRISE separately. Selected fellows work on projects with Harvard-affiliated researchers and get to live in the Harvard Summer Undergraduate Research Village (HSURV) with fellows in the PRISE, BLISS, PRIMO, SHARP, SURGH, and PCER research programs. These fellows live in one of the Harvard College houses and participate in extremely rich evening and weekend programming that includes both social and academic activities. In addition to receiving free lodging and being members of a diverse, vibrant intellectual and social community, fellows also receive a nominal stipend, partial board, and (for those students on financial aid) full coverage of the required summer savings obligation.

Harvard-Amgen Scholars Program is a ten-week residential summer research program for undergraduates in biotechnology. Following an interview selection process, participants in the Harvard-Amgen Scholars Program will be paired with faculty mentors and a direct supervisor (postdoctoral fellow or graduate student) in the laboratory. In addition to research, Amgen Scholars will participate in a number of intellectual, pre-professional development, and social activities throughout the program.

Harvard-Amgen Scholars live in one of Harvard's historic houses as active members of the Harvard Summer Undergraduate Research Village community. The cohort will comprise both Harvard students and students from other US institutions. Harvard-Amgen Scholars also receive a generous stipend, roundtrip travel to the Amgen Scholars Symposium in Los Angeles, CA, and (for those students on financial aid) full coverage of summer saving obligation.

Applicants must meet the following criteria: US citizenship or US permanent residency status, enrolled in good standing as a current sophomore, junior, or non-graduating senior, with a cumulative GPA of 3.2 or above.

Herchel Smith is a competitive and generous award supporting high potential undergraduates who are conducting promising summer research projects in mathematics; engineering; and life, physical, natural, or computer sciences. The program experience is designed specifically to prepare students in good standing for competitive postgraduate fellowships (such as NSF), graduate/doctoral study domestically and internationally (such as at Cambridge University), and post-baccalaureate research positions in the private sector. The project can be based anywhere in the world, but must be affiliated with a university, lab, or research enterprise and be highly substantive: at least ten weeks in duration, full-time in commitment, and exhibiting some degree of autonomy and input by the applicant in its design and execution.

HCRP provides funding in support of student-initiated, independent scholarly research or creative endeavors undertaken with guidance of Harvard-affiliated faculty mentors. HCRP grants advance academic experiences outside the classroom and expand opportunities for students to work closely with faculty members. In contrast to a research assistantship, HCRP recipients demonstrate autonomy in the development, direction, and preparation of the overall research project. Awards are available for fall and spring terms of the academic year, as well as for the summer. Undergraduate students from all concentrations are encouraged to apply.

Suggestions for Other Sources of Research Funding:
1. Visit CARAT (Centralized Application for Research and Travel) to identify funding sources to which you can apply for term-time or summer: **https://carat.fas.harvard.edu/**.

2. Check with your academic department to inquire whether there are any funds to support students conducting research. Some departments may have funds to help a student present a paper at a conference or conduct senior thesis research.

3. Visit the Student Employment Office (SEO) Jobs Database to look for posted research assistant positions: **https://seo.harvard.edu/**.

4. Talk with your faculty advisor to see if they would be willing to apply for the Faculty Aide Program (FAP). FAP encourages professors to hire undergraduate research assistants by subsidizing students' pay. Potential faculty hosts are encouraged to post these research assistantships through the SEO's jobs database. Note: while faculty members apply for this award, students are encouraged to inform their faculty mentors about FAP. Knowing that FAP can subsidize students' pay often increases the ability/willingness of faculty to hire student researchers on their projects. For more information visit **https://seo.harvard.edu/faculty-aide-program.**
Contact: Pamela Gaddi, pjgaddi@fas.harvard.edu, uraf.harvard.edu
77 Dunster Street, 2nd floor, Cambridge, MA 02138

Table 5. Mind Brain Behavior
As an interfaculty initiative, Mind/Brain/Behavior (MBB) does not have its own faculty and research programs as departments do. Instead, it serves as a clearinghouse for research opportunities for undergraduates. These opportunities are available to undergraduates in MBB tracks and the MBB secondary field but also often to students who are not in a formal MBB program but who have interests in mind, brain, and/or behavior. MBB students planning to do thesis research the summer before their senior year may apply for Mary Gordon Roberts MBB Summer Thesis Research Fellowships to help fund their work.
Contact: Shawn Harriman, shawn_harriman@harvard.edu, https://mbb.harvard.edu/pages/undergraduate-research
1384 William James Hall, 33 Kirkland Street

Table 6. Harvard College Undergraduate Research Association
We are a student-run organization dedicated to fostering a research community at Harvard. We run the largest research conference for undergraduates in the country, and we connect students to professors and industry research opportunities.
Contact: Connie Cai, ccai@college.harvard.edu, https://www.hcura.org/
121 Cabot Mail Center 60 Linnean Street Cambridge, MA 02138

Table 7. Radcliffe Research Partnership
Harvard College students can work one-on-one or in a small group on a project supervised by a Radcliffe fellow in the natural sciences and mathematics. Students are paid $15.00/hr and may receive mentoring from accomplished researchers. During the summer, these paid internships may be one month long (June or July) or may span two months (June through July). Students are encouraged to contribute to

the project in significant ways. Past participants have found the program to be fulfilling as well as flexible.
Contact: Jess Yoo, rrp@radcliffe.harvard.edu,
https://www.radcliffe.harvard.edu/fellowship-program/radcliffe-research-partnership
Radcliffe Fellowship Program 8 Garden Street Cambridge, MA 02138

Table 8. MGHfC Digestive Disease Summer Research Program
The MGHfC Digestive Disease Summer Research Program offers short-term NIH support for 10 students matched with research mentors to perform independent research focused on digestive diseases over a 10-week period during the summer. In addition to presenting their research at a summer's end symposium, students will also participate in a peer-driven presentation-based biomedical course and a panel discussion on biomedical careers. Currently 22 MGH independent investigators with interest in digestive diseases serve as mentors spanning a wide range of topics and technical training. Research topics include obesity, food allergy, microbial pathogenesis, gut-brain axis, probiotics, intestinal development and maintenance, cystic fibrosis, celiac disease, inflammation, intestinal parasites, and adaptive immunity. Techniques applied to research projects include, advanced imaging, microfluidics, molecular biology, computational biology, stem cell biology, immune cell isolation, in vivo modeling, and patient-based clinical research. The MGHfC Digestive Disease Summer Research Program provides an excellent opportunity for science, math, and engineering students interested in biomedical research.
Contact: Bryan Hurley, bphurley@partners.org,
https://www.massgeneral.org/mucosal-immunology/Education/summer-research-program.aspx
Pediatrics - Mucosal Immunology & Biology Research Center
Massachusetts General Hospital 55 Fruit Street, Jackson 1402 Boston, MA 02114

Table 9. Office of Career Services
Students who find research assistant positions abroad that are 8+ weeks can apply to our office for funding.
Contact: Leanne Gaffney, leanne_gaffney@harvard.edu,
https://ocs.fas.harvard.edu/summer-funding
54 Dunster Street, Cambridge, MA 02138

Table 10A. Harvard Forest Summer Research program

The Harvard Forest Summer Research Program, based at Harvard's 4,000-acre laboratory and classroom in Petersham, Mass., is an REU-based, paid internship program that brings 20-30 students from across the US to live and work at the Forest for 11 weeks. Interns work with a research mentor on interdisciplinary and often team-based research projects focused on how ecosystems change over time, especially in response to human and natural disturbance. Program seminars, field trips, and professional development workshops in data analysis and science communication build on students' individual research projects and help them explore career and graduate school options. The 2019 program dates are Monday, May 27 - Friday, August 9, 2019. Students apply to specific projects, which range from community ecology and history to soil science, atmospheric chemistry, science communication, landscape modeling, and data provenance. Projects will be posted on the Harvard Forest website in early December. Applications are due February 1, 2019. Harvard undergraduates from all concentrations are encouraged to apply.
Contact name: Manisha Patel, Program Coordinator; Audrey Barker Plotkin, REU Site PI
Address: 324 North Main Street, Petersham MA Tel: 978-724-3302
Email: manishapatel@fas.harvard.edu or aabarker@fas.harvard.edu
Website: https://harvardforest.fas.harvard.edu/other-tags/reu

Table 10B. Biological Sciences in Public Health PhD Program (BPH)
The Ph.D. Program in Biological Sciences in Public Health provides students with interdisciplinary training in cutting-edge biomedical research on mechanisms influencing human health and widespread diseases, translating these scientific insights into practices that improve global public health. Students wishing to do research in areas of metabolic dysregulation in complex disease (obesity, metabolic syndrome, cancer), health effects of environmental exposures (air pollution, lung infection, asthma), nutritional biochemistry (nutrient transport and metabolism), gene-environment interactions (epigenetics, inflammation, stress response), immunology and infectious diseases (host-pathogen interactions and protozoa, helminths, viruses or bacteria) usually study in the Biological Sciences in Public Health (BPH) program. Participating HSPH departments - Genetics and Complex Diseases, Environmental Health, Immunology and Infectious Diseases, Nutrition Summer Internship Program in Biological Sciences This intensive 8 week laboratory-based biological research program is for undergraduates during the summer following their sophomore or junior years. Application opens up early Nov.

Contact: Tom Brazda, tbrazda@hsph.harvard.edu,
https://www.hsph.harvard.edu/biological-sciences/
655 Huntington Avenue, Building 2, Room 119, Boston, MA 02115

Acknowledgments

HUROS Organizers

Anna Babakhanyan, PhD, Science Undergraduate Research Advisor
Margaret A. Lynch, PhD, Associate Director of Science Education



The Science Education Team

Logan McCarty, PhD, Director of Science Education
Irina Cashen
Ricardo Mora

James Poolner
Leigh Stimolo

Office of Undergraduate Research and Fellowships

Greg Llacer
Christopher Kabacinski



Andrea Lamas-Nino
Connie Cai
Michael Xie
Molly Zeme

The Harvard name and/or VERITAS shield are trademarks of the President and Fellows of Harvard College and


HARVARD UNIVERSITY
science
education


HARVARD COLLEGE
Office of Undergraduate Research and Fellowships


HARVARD COLLEGE UNDERGRADUATE
RESEARCH ASSOCIATION