IBS Lab Rotation List

Fall 2015 [September 14th – December 4th]
Spring 2016 [January 4th – March 25th]
Summer 2016 [March 28th – June 3rd]

Dr. Brindley:    Availability: □ Fall 15  ☑ Spring 16  ☑ Summer 16
Neglected Tropical Diseases, and Helminth infection induced cancers.

Please see lab website,

http://smhs.gwu.edu/brindley-lab/

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Dr. Bukrinsky:    Availability: □ Fall 15  ☑ Spring 16  □ Summer 16
We are studying function and mechanism of action of HIV-1 accessory proteins Nef and Vpr. These proteins are responsible for many pathogenic effects of HIV infection, and our studies seek to characterize interaction of Nef and Vpr with host cell proteins.

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Dr. Caldovic:    Availability: □ Fall 15  ☑ Spring 16  ☑ Summer 16
My research focuses on finding new treatments for hyperammonemina (elevated blood ammonia), which can be due to inborn errors of metabolism or liver failure. Ammonia is a potent neurotoxin that causes permanent brain damage. I am using zebrafish and mouse models of hyperammonemia to screen for drugs that can protect the brain from ammonia toxicity.

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**Dr. Chen:**

**Availability:**  ☑ Fall 15  ☑ Spring 16  ☑ Summer 16

We investigate molecular mechanisms involved in genetic diseases. After potential therapeutic targets are identified, we conduct in vitro assay and preclinical studies to identify and validate potential treatments for the diseases. We are also involved in clinical studies with a focus on biomarker identification.

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**Dr. Colonnese:**

**Availability:**  ☑ Fall 15  ☑ Spring 16  ☑ Summer 16

My lab studies the synaptic, network and metabolic changes that allow the developing brain to first begin to process information. We use the development of vision as a model system to understand when and how the cerebral cortex first comes “online” to engage in conscious and sub-conscious processing of external stimuli. This provides testable hypotheses about the origins of consciousness in the fetus and has implications for treatment of disrupted cognition in neurodevelopmental disorders.

Our immediate questions are (1) when are humans and other mammals first capable of sight, (2) what are the critical developmental checkpoints leading to sight and thought, and (3) why do these process occur when they do? We answer these questions by assaying the ensemble activity of neural circuits using state of the art electrophysiological techniques, including multi-electrode arrays and intra-cellular recording, combined with optogenetic and transgenic manipulation of circuit dynamics, in behaving neonatal rodents. By comparing our recordings with EEG recordings of human preterm infants gathered by collaborators, we make predictions about the relevance to human fetal and perinatal development.

Potential thesis projects include determining the neuromodulatory control of the development of wakefulness, characterizing cortical activity patterns during critical periods for plasticity, analysis of human preterm EEG for homology with rodent brain activity, determining the cortical circuit defects in mouse models of human neurological disorders, understanding and treating cortical activity defects associated with preterm birth, and computational analysis of multi-electrode recordings. Rotation projects will provide hands-on experience in surgical, anatomical and electrophysiological techniques during investigation of a small self-contained question designed in consultation with the student.

Students interested in the ongoing direction and techniques of the laboratory should review the following papers:


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**Dr. Corbin:**

The Corbin lab studies developmental genetic mechanisms underlying formation of limbic system circuitry and innate behaviors.

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**Dr. Crandall:**

The Crandall Lab is a computational biology/bioinformatics lab that develops and tests methods of analysis in phylogenetics and metagenomics. We apply such methodology to a wide variety of questions in infectious disease. If you are looking for a rotation centered on informatics approaches and data analysis, this is the place. We are housed in the Computational Biology Institute (cbi.gwu.edu) where you will interact with a variety of faculty working on a diversity of projects centered in computational biology.

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**Dr. Freishtat:**

I am the principal investigator of The AsthMaP™ Project. My current research interests include systems biology investigations of injury/repair in the lung with particular focus on airway epithelial stem and progenitor cell proliferation in asthma.

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Dr. Hashimoto-Torii:  Availability: Fall 15  Spring 16  Summer 16

Exposure of human fetuses to a multitude of physical and chemical environmental stressors causes a wide variety of abnormalities with serious health consequences. The embryonic central nervous system is highly vulnerable to environmental exposures, as it displays not only overt cytological malformations, but also a covert increase in the susceptibility to late-onset neuropsychiatric disorders. The goal of Hashimoto-Torii laboratory is to understand how adverse prenatal environment interacts with genetic predisposition, thereby increasing the disease susceptibility after birth. With a focus on the cerebral cortex, we tackle this challenging question by a combination of wet and dry analyses.

Wet Lab: Contribution of Stress Responsive Genes to Increased Susceptibility to Neuropsychiatric Disorders. We recently found that differential strength of cellular stress responses among the cortical cells leads to different cellular phenotypes in response to the adverse prenatal environment. Therefore, understanding how cells in close proximity show different responses to the same stresses will provide critical insights into the etiology of prenatal environment-initiated neuropsychiatric disorders. Using state-of-the-art imaging techniques and our house made reporter transgenic mice, we aim to answer this question.

In another line of our research, we have obtained a list of genes that are expressed in specific types of neural cells and respond to prenatal environmental challenges. We will examine the roles of these genes in normal brain development through loss/gain of function analyses and other molecular/biochemical/cell biology approaches.

Dry lab: Epigenomic Profiling of Prenatal Environmental Effects on Brain Development. Human fetal cerebrum, which is particularly sensitive to physical and chemical agents due to a much more prolonged period of neurogenesis as well as longer distance neuronal migration and protracted differentiation. Magnetic resonance imaging (MRI) and postmortem examinations of the human brain reveal various consequences of fetal adverse environment, but only long after initial exposure to harmful agents. Most of our knowledge about the cellular and molecular mechanisms of brain damage caused by adverse environment, therefore, has been gained from experimental studies in rodents. In order to fill these gaps between human and animal studies, we established collection of human fetal brain specimens which had been exposed to environmental stressors, such as maternal epilepsy, infection, inflammation, drinking and medication which all have been known to affect the offspring’s behaviors. Using this unique collection, we perform a battery of epigenomic profiling. Questions are; which brain regions or neural cell types are more vulnerable against environment than others at epigenomics level, and whether classification of the environmental stressors based on the epigenomic profiling matches with that based on the induced phenotypes.

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Muscular dystrophies and Duchenne muscular dystrophy in particular are debilitating diseases that affect children worldwide. Duchenne is due to missense mutations in the dystrophin gene leading to complete loss of expression of the dystrophin protein that is essential to muscle fiber integrity and function. Clinically, DMD is characterized by progressive muscle degeneration leading to loss of ambulation by 8-12 years of age and death by early adulthood due to cardiorespiratory failure. There is no effective curee for DMD currently. Chronic high dose glucocorticoids are considered standard of care. Their use increases muscle strength and function and can delay loss of ambulation by 1 to 2 years but concerns about severe side effects often restricts their use. New promising therapeutic strategies have been tested in animal models and are now entering clinical trials. However moving forward with these treatments in human has been slow due to the lack of outcome measure to determine efficacy. In my laboratory at the Center for Genetic Medicine at Children’s National Medical Center we are interested in the following areas of research:

- Define new therapeutic targets for the treatment of Duchenne by studying the molecular mechanism at early stage of the disease.
- Develop serum biomarkers to monitor disease progression and response to therapies when using new drugs
- Evaluate efficacy and safety of new treatments

Materials and methods to be used are:

**Materials:** Clinical samples collected from patients (serum and muscle tissue), mouse models and muscle cell cultures to test new drugs.

**Methods:** Tissue dissection and fractionation, cell fractionation, Proteomics (protein separation methods, protein quantification methods, gel electrophoresis, chromatography, mass spectrometry, bioinformatics), histology (tissue micro dissection, microscopy).

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Dr. Horvath:  

Availability: Fall 15 ☑ Spring 16 ☑ Summer 16

Our research program has two main aspects. The first is focused on identifying biologically significant findings from Next Generation Sequencing (NGS) datasets and developing novel strategies to link genetic patterns to phenotype characteristics, and to regulatory trends. The rapid development of NGS technologies has resulted in the exponential growth of scientific data that provide unique insight into fundamental cellular processes and disease conditions. However, a major challenge is the logical mining of high-throughput data to extract meaningful set of high-priority molecules. We apply computational genomics strategies to develop pipelines that efficiently integrate the standard alignment, assembly, and variants analysis, with custom analytical modules based on the project specifics. In addition, we design and develop novel algorithms to align genomic and transcriptomics information from the same sample(s), in search for links between encoded and regulatory patterns, and for co-existing, or mutually exclusive features.

The second aspect of our research deals with applications of the above methodologies on breast cancer. We apply the developed analytical pipelines to search for pathogenic and protective genetic patterns in breast cancer. We interlink essential NGS outputs, such as allelic loss and splice-modulating potential in NMD-suppressed setting (NMD, Non-sense mediated mRNA decay), to outline functional molecular networks that can be further implemented in diagnostic and preventive strategies.

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Dr. Khan:  

Availability: Fall 15 ☑ Spring 16 ☑ Summer 16

The research focus of my laboratory is to study the development and maintenance of CD8+ T cell memory against infectious agents like Toxoplasma gondii and Encephalitozoon cuniculi infection. Both of these pathogens are opportunistic infections that pose severe risks to HIV infected population. Maintenance of effective CD8 T cell immunity in an immune-compromised situation is a challenge and the laboratory is interested to develop strategies the can ensure the persistence of protective immunity.

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Dr. LaMantia:     Availability: ☒ Fall 15 ☒ Spring 16 ☒ Summer 16

Two projects are available: The first deals with development of cortical layer 2/3 circuitry in a mouse model of 22q11Deletion Syndrome, a genetic disorder that results in autism, ADHD and/or schizophrenia-like behavioral disorders. The second deals with the specification of olfactory neural stem cells: the only neural stem cells retained in humans that generate new neurons throughout life.

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Dr. Liu:     Availability: ☐ Fall 15 ☒ Spring 16 ☒ Summer 16

Progress toward developing effective treatments for severe therapy-resistant asthma (STRA) has been limited by the inadequacy of existing preclinical animal models of asthma. As an alternative, xenograft models have proven informative with regard to human airway diseases that are difficult to replicate in animals. Using xenograft techniques, we have established a true animal model of human asthma, with replication of both asthmatic airway inflammation and remodeling simultaneously. These xenografts are comprised of human asthmatic airway epithelium grown on a matrix of decellularized rat trachea implanted subcutaneously in nude mice. This unique model of asthma is well-suited for preclinical trials, especially with regard to predicting individual responses to drug.

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Dr. Mann:     Availability: ☐ Fall 15 ☒ Spring 16 ☒ Summer 16

Neglected Tropical Diseases, and Helminth infection induced cancers.

Please see lab website,

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Dr. Manzini: 

The goal of my lab is to study the determinants of neuronal differentiation and circuit formation in the normal and diseased brain. We start from the identification of human genes, which are essential for normal cognition and cause neurodevelopmental disorders ranging from autism to severe brain malformations. We then use animal models (mouse and zebrafish) to recapitulate the disease state, understand pathogenesis and study the molecular mechanisms of development. For more details please visit our website www.manzinilab.org

There are currently two projects open for graduate students in the lab.

1) Genetic causes of dystroglycanopathies
   The goal of this project is to use next-generation sequencing (exome, genome and RNA-seq) to identify novel disease mutations causing the most severe forms of congenital muscular dystrophy. These disorders are characterized by muscular, ocular and brain malformations, which can be recapitulated in zebrafish models. The project will entail dealing with human samples for preparation of different sequencing libraries, data analysis, gene identification and using the zebrafish as a model using morpholino oligonucleotides or genome engineering (CRISPR/Cas). It requires a highly organized, independent and enthusiastic individual who will learn how to handle large datasets and bioinformatic tools as well as molecular biology techniques. The rotation will focus on establishing CRISPR/Cas approaches in the lab.

2) Intracellular signaling regulation of cognitive development
   The goal of the second project is to study the role of intracellular signaling regulation in the etiology of intellectual disability (ID) and autism spectrum disorder (ASD). We have identified a gene, CC2D1A, which when mutated in humans causes a spectrum of ID and ASD. We have developed animal models of Cc2d1a loss of function and are characterizing them at the molecular, cellular and behavioral level. There are multiple state-of-the-art cellular imaging approaches being developed in the lab and a full set of behavioral paradigms. This project requires a skilled, creative and motivated team player who can work with two postdocs and who is willing to develop an independent project. The rotation will focus on imaging dendritic spines and synapses during development in wild-type and human mice.

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Dr. Mendelowitz:

Our research is focused on the autonomic and respiratory control of brainstem cardiovascular function in both normal physiological homeostasis as well as alterations that occur to initiate and/or sustain cardiorespiratory diseases. In particular we study the cellular properties and neuronal network and reflex control of pre-motor parasympathetic cardio-inhibitory vagal neurons located in the nucleus ambiguous in the brainstem. The activity of these cardiac vagal neurons dominates the neural control of heart rate, yet despite their clinical importance we have
only begun to fully understand the transmitters and integration of complex synaptic pathways from other brain sites that control these critical specialized neurons. We explore how the brainstem generates the parasympathetic control of heart rate in healthy subjects, and the changes that occur in disease states such as Sudden Infant Death Syndrome (SIDS) and Obstructive Sleep Apnea (OSA). Approaches include combinations of whole cell patch clamp electrophysiology, viral tracing, transgenic animal models, photoexcitation using optogenetics (channelrhodopsin) and UV-uncaging, and whole animal telemetry recordings of blood pressure and heart rate.

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Dr. Miller: Availability: ☐ Fall 15 ☑ Spring 16 ☑ Summer 16

Dr. Miller has a primary interest in CNS neural development with a focus on understanding the biology of neural diseases including Multiple Sclerosis, Brain tumors and Cerebral Palsy. Current programs in the Miller lab are targeted at developing new therapies to promote CNS repair in Multiple Sclerosis. These studies include the use of cellular therapies and molecular approaches to enhance oligodendrocyte development, differentiation and myelin repair.

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Dr. Nazarian: Availability: ☐ Fall 15 ☑ Spring 16 ☑ Summer 16

Our laboratory works on molecular mechanism of disease progression in pediatric brain tumors. Specifically, we work on proteomics, genomics and methylation patterns of these tumors. Our goal is to use our in vivo and in vitro models to develop therapeutics for treating DIPGs.

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Dr. Penn: Availability: ☐ Fall 15 ☑ Spring 16 ☑ Summer 16

Neuropentractology: linking placental function to brain development and damage. Our laboratory's goal is to understand the hormonal factors that contribute to normal development and the impact of their loss following premature birth or placental compromise. Many events, including infection, malnutrition, and genetic abnormalities can disrupt the placenta's function, or – as in preterm birth – can abruptly change the brain’s hormonal environment. Such changes
may directly damage the developing brain or increase its susceptibility to the damage that leads to cerebral palsy or developmental delay. Our long-term objective is to develop novel neuroprotective replacement therapies using a multi-faceted approach. Using lentiviral RNA suppression specifically in the placenta, conditional knockout techniques and rodents models of preterm brain injury, we are testing these hormonal neurodevelopmental mechanisms at the cellular, anatomical and behavioral level.

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**Dr. Sen:**

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Availability: Fall 15, Spring 16, Summer 16

I am currently funded by American Heart Association title: “Use of P53 silenced EPC to treat ischemia in diabetic peripheral vascular disease, and Astra-Zeneca & Am Diabetes Association- Investigator Initiated Study, title” Use of CD34+ cell number, function and gene expression in evaluating endothelial dysfunction in patients with early type 2 diabetes, pre and post saxagliptin therapy.
My third project is funded by Bohringer-Ingelheim & Am Diabetes Association- Investigator Initiated Study, titled,"Role of linagliptin in improving renal failure by improving CD34+ stem cell number, function and gene expression in renal function impaired type 2 diabetes patients.
The fourth project is based on cell metabolism titled, Use of genetically engineered human adipose tissue derived mesenchymal stem cells (MSCs) to reduce inflammation in diabetes and obesity". Our lab is interested in using stem cells as it is (de-novo) and also post manipulation using a non-integrating DNA virus such as Adeno or Adeno-associated virus to upregulate or silence a gene of interest in stem cells

**Dr. Seto:**

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Availability: Fall 15, Spring 16, Summer 16

The major interest of my laboratory is in the area of cancer epigenetics, more specifically on the functions, mechanisms of action, and regulations of histone deacetylases (HDACs). In addition to our work on HDACs in transcriptional co-repressor complexes, we discovered that HDACs regulate important biological processes beyond histone modification, such as cell growth, proliferation and repair. For example, we found that SIRT1 deacetylases and regulates the function f the NSB1 protein, and that the Class II deacetylase HDAC6 modifies cortactin and regulates cell movement. The long-term objective of our research is gain insight into the novel functions and mechanisms of action of HDACs, which will lead to potential diagnostic and therapeutic approaches for patients affected with HDAC-relevant cancers.
Contact:
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Dr. Shanmugam:  Availability: ☒ Fall 15 ☒ Spring 16 ☒ Summer 16

The Shanmugam Laboratory takes a multidisciplinary, collaborative approach to the scientific process. Inclusion of clinicians, nurse scientists, biotechnology experts and post-doctoral scientists allows us to develop synergistic projects that advance scientific discovery and translate discoveries into action. By focusing on clinical observations, research projects in the Shanmugam Laboratory are always centered on clinically important aspects of the disease process which drastically increases our yield for identifying important molecular drivers and novel therapeutic targets of disease. The Shanmugam Laboratory currently has an IBC approved research protocol and three IRB approved studies with a focus on scleroderma and wound healing.

The Wound Etiology and Healing (WE-HEAL) Study (IRB: 041408) is a biorepository dedicated to studying the wound healing process. The WE-HEAL Study examines how the immune system, the wound microbiome, and pain pathways relate to wound healing. In the lab, students will learn keratinocyte culture methodology as well as experiments such as scratch assays or flow cytometry. Students will also learn how to process and biobank biospecimens such as blood, serum, wound fluid, tissue, and biofilm. They will learn RNA isolation, PCR and RT-PCR techniques as well as gene expression analysis. Students will also learn how to perform ELISA multiplex assays on wound effluent and serum.

The Scleroderma Biorepository and Pathogenesis Study (STOP Scleroderma) (IRB: 051427) investigates why patients develop certain complications from scleroderma. We collaborate with multi-center studies which focus on early diffuse disease and the genomics of scleroderma in the African Americans. In the lab, students will learn DNA isolation and quantification techniques. They will also learn how to isolate lymphocytes, plasma, and serum from blood.

In conclusion, The Shanmugam Laboratory will expose students performing lab rotations or dissertations to a variety of lab techniques in the field of immunology, biochemistry, and biology while unlocking the secrets to the wound healing process and scleroderma.

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Dr. Sotomayor:  Availability: ☐ Fall 15 ☒ Spring 16 ☒ Summer 16

My research focuses on lymphoma and immunotherapy. More specifically, our work pertains to immunotherapy of B-cell malignancies, with special emphasis on the design of novel immunotherapeutic approaches for these diseases. My laboratory has an extensive portfolio of basic, translational, and clinical research initiative for mantle cell lymphoma, an aggressive subtype of B-cell non-Hodgkin lymphoma. We investigate how the information in immune cells can be used to make them better at recognizing tumor cells.
Dr. Stepp:  
We study wound healing using the mouse cornea as a model system. Projects involve studies of reinnervation of sensory axons after injury and maintenance of the stem cell population at the limbus. The potential exists for collaborations with groups from the NIH who study epidermal differentiation and skin cancer.

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Dr. Torii:  
The cerebral cortex consists of a large variety of neuronal and glial subtypes with specific morphological and functional characteristics associated with distinct molecular properties. During development, cortical neurons and glia migrate from their sites of origin to their specific final locations within the laminar and columnar organization of the cerebral cortex, and form stereotypical connections within the cerebral cortex, as well as with cells in other parts of the brain.

The goal of our lab is to elucidate the molecular and cellular mechanisms that govern unique positioning and connections of various neuronal and glial subtypes in normal cortical development, and understand the etiology of cognitive and psychiatric disorders in which abnormalities in these processes may be involved. Toward this goal, we use a variety of tools and techniques including in vivo gene manipulation, transgenic mice, human tissue specimens, cell lineage and neural circuit tracing, and time-lapse live imaging of cells.

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Dr. Triplett:  
The human brain contains approximately 100 billion neurons each of which can make and receive connections to and from over 1000 neurons. In order to perform the complex behaviors that make us who we are, synaptic connections must be made with a high degree of specificity. Indeed, disruptions in wiring can result in an array of neurodevelopmental disorders, such as autism, ADHD and schizophrenia. The goal of my lab is to understand the molecular and
activity-dependent mechanisms that regulate the precise development of neuronal connections. Towards this audacious goal, we focus on the development of sensory circuits in a midbrain structure, the superior colliculus (SC), which integrates visual, somatosensory and auditory information to direct head and eye movements. In wiring the SC, these converging inputs must accomplish two critical tasks. First, they must preserve the spatial order of the outside world my establishing and aligning topographic maps in each modality. Second, they must preserve qualitative channels of sensory information, i.e. visual neurons in the eye tuned to movement vs. contrast must connect with the corresponding neurons in the SC. However, the mechanisms by which these amazing wiring feats are established remains poorly understood. To investigate this problem, the Triplett lab employs a variety of techniques in transgenic and genetic knock-out model organisms. Antero- and retrograde axon tracing reveals the anatomical organization of neuronal connections, fluorescence activated cell sorting followed by microarray analysis identifies molecular candidates required for proper guidance, and in vivo electrophysiology is used to determine the functional properties of neurons. Identifying the cues required for establish precise connectivity and functionality could have broad-reaching impacts on developmental disorders, such as autism, schizophrenia and sensory processing disorders. followed by microarray analysis identifies molecular candidates required for proper guidance, and in vivo electrophysiology is used to determine the functional properties of neurons. Identifying the cues required for establish precise connectivity and functionality could have broad-reaching impacts on developmental disorders, such as autism, schizophrenia and sensory processing disorders.

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Dr. Tyagi:  
Availability:  ⚫ Fall 15  ⚫ Spring 16  ⚫ Summer 16

We work on various aspects of HIV focusing mainly on HIV latency, the major hurdle in HIV eradication. We investigate the various molecular and epigenetic mechanisms which regulate the latent state of HIV in primary CD4+ T cells. In order to study HIV latency we have developed a model system for HIV latency in primary CD4+ T cells. We study the underlying molecular mechanisms that are modulated by cocaine in order to enhance HIV replication. We also analyze how co-infections such as HCV to HIV patients affect HIV and HCV life cycle. Recently, we have also started the role of HIV and HCV on aging process; as HIV infected population acquires age related diseases 10 to 20 years earlier than uninfected individuals.

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Dr. Tzatsos:  
**Availability:** ☐ Fall 15 ☑ Spring 16 ☑ Summer 16

Cancer epigenetics and mouse modeling.

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Dr. Wu:  
**Availability:** ☑ Fall 15 ☑ Spring 16 ☑ Summer 16

Epigenetics plays an important role in human health and disease. Disruption of epigenetic modifications leads to dysregulation of gene function without altering the DNA sequence per se. A major challenge in understanding the importance of epigenetics in human health and diseases is to identify the specific epigenetic factors and the signaling involved.

One of the projects in my lab is to study the role of two epigenetic modulators, ARID4A and ARID4B, members of the AT-rich interaction domain family in development and disease. Currently, my lab uses state-of-the-art conditional knockout mouse and transgenic mouse models to investigate the normal physiological and pathophysiological functions of ARID4A and ARID4B, and genomic and proteomic approaches to identify the molecular mechanisms underlying their action.

**References:**


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Dr. Xia Zheng:  
**Availability:** ☑ Fall 15 ☑ Spring 16 ☑ Summer 16

The Hedgehog (Hh) signaling pathway organizes pattern formation in a variety of embryonic tissues and functions post-embryonically in homeostatic processes. Hh pathway dysfunction thus can lead to embryonic pattern disruptions, such as holoprosencephaly and other birth defects in humans; post-embryonic dysfunction can result in failure of adult tissue regeneration as well as proliferative disorders, such as cancer.

My primary research interest is in identifying target genes regulated by the Hh signal and thus to understand the molecular mechanisms employed by the Hh signaling pathway in regulating cell-
cell interactions (I). In parallel, I am interested in developing novel reagents and experimental approaches combined with cutting-edge imaging technologies to study the biochemical and cell biological principles governing a critical yet poorly understood step of Hh signal transduction: trafficking of Hh receptors (II). These studies have broad potential significance for our understanding of the molecular basis of both development and human diseases linked to Hh pathway dysfunction, as well as for providing a basis for therapeutic modulation of pathway activity, either positively to stimulate regeneration or negatively to block malignant growth.

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**Dr. Pan Zheng:**

My lab works on signal transduction in hematopoiesis and T cell development. We focus on TSC-mTOR and Wnt signaling pathways. We have several exciting projects in aging, inflammation and cancer immunology.
http://childrensnational.org/research-and-education/about-cri/faculty-and-leadership-directory/pan-zheng

**Availability:** ☑ Fall 15  ☑ Spring 16  ☑ Summer 16

**Dr. Zhu:**

Dr. Zhu’s research group is interested in understanding molecular and cellular mechanisms underlying the development of normal neural stem and progenitor cells as well as tumorigenesis in the nervous system. We are using the mouse as a model system to develop genetic engineering mouse (GEM) tumor models, which recapitulate human nervous system tumors both genetically and phenotypically (Zhu et al., Cell 1998; Zhu et al., Science 2002; Zhu et al., Cancer Cell 2005; Zheng et al., Cancer Cell 2008; Wang et al., Cancer Cell 2009; Wang et al., Cell 2012). Particularly, we have been focused on the role of tumor suppressor genes in the nervous system.

**Neurofibromatosis type 1 (NF1):** In addition to the high risk of developing tumors in the nervous system, approximately 30-70% of individuals with NF1 have learning disabilities, representing the most significant cause of lifetime morbidity associated with this disease. Dr. Zhu’s research group is interested in understanding the role of Nf1 in developing neural stem and progenitor cells and how its loss causes developmental abnormalities, leading to the structural brain defects associated with severe learning disabilities in humans, and the development of benign peripheral nerve sheath tumor – plexiform neurofibroma in the peripheral nervous system (PNS) and optic pathway glioma in the central nervous system (CNS). Dr. Zhu’s research group is investigating the mechanism underlying these NF1-associated diseases and performing
preclinical studies with animal models. Our goal is to integrate basic, translational and clinical research to develop novel preventive and treatment therapies for NF1-associated diseases.

High-grade glioma and glioblastoma (GBM): NF1 tumor suppressor gene is one of the most frequently mutated genes in GBM – the most frequent and lethal brain cancer in humans. However, the development of GBM in individuals afflicted with NF1 is not common. Using GEM models, we have demonstrated that inactivation of Nf1 is not a robust oncogenic event unless it occurs in the context of p53 loss. Thus, sequential inactivation of tumor suppressor genes p53 and Nf1 is required for effectively transforming neural stem and progenitor cells in the subventricular zone (SVZ) of the lateral ventricle. These studies have established NF1 as a context-dependent tumor suppressor gene in GBM, providing the mechanism by which most individuals with NF1 have no increased risk of developing GBM. We are exploring these mouse models to address: (1) how the tumor suppressor genes p53 and Nf1 regulate growth and transformation of neural stem/progenitor cells in vivo and in vitro, (2) what is the lineage relationship between neural stem and progenitor cells or differentiated cells and tumors in the nervous system, and (3) what is the molecular mechanism(s) underlying the development of astrocytomas/GBM and malignant peripheral nerve sheath tumors (MPNSTs) from normal neural stem and progenitor cells.

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**Dr. Zohn:**
Availability: ☑ Fall 15 ☑ Spring 16 ☑ Summer 16

The research in my laboratory focuses on understanding the cellular and molecular basis of structural birth defects such as neural tube (spina bifida) and heart defects. We utilize a variety of strategies and mouse models in addition to human genetic studies.

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