IBS Lab Rotation List

Fall 2013 [September 16th – December 6th]
Spring 2014 [January 6th – March 28th]
Summer 2014 [March 31st – June 6th]

Dr. Berl:  
Availability: ☑ Fall 13 ☑ Spring 14 ☑ Summer 14

We utilize neuroimaging (fMRI, DTI, T1, T2) and neuropsychological studies to investigate the neural underpinnings of cognition in children with epilepsy including how functions are reorganized and/or perturbed due to seizures and/or treatments.

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Dr. Brindley:  
Availability: ☑ Fall 13 ☑ Spring 14 ☐ Summer 14

*Neglected Tropical Diseases  
*Studies with transgenesis and vector mediated RNA interference in schistosomes  
*Infection-related cancers: molecular pathogenesis of helminth parasite induced tumors

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Dr. Bukrinsky:  
Availability: ☑ Fall 13 ☑ Spring 14 ☐ Summer 14

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 13 ☑ Spring 14 ☑ Summer 14

My research focuses on finding new treatments for hyperammonemia: 1) Screening for drugs that protect the brain from the toxic effects of ammonia; and 2) Increasing expression of defective enzymes in patients with inborn errors of protein catabolism.
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Dr. Chen: Availability: ☑ Fall 13 ☑ Spring 14 ☑ Summer 14

We are interested in studying molecular mechanisms involved in muscle adaptation and disorders using both cellular and animal models. Genes and pathways identified that are identified to be important to disease mechanisms are further studied as therapeutic targets. Treatment development for facioscapulohumeral muscular dystrophy (FSHD) and Duchenne muscular dystrophy (DMD) using genetic and pharmaceutical strategies are ongoing. Additionally, we develop transgenic mouse models for testing these approaches.

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Dr. Colberg-Poley Availability: ☑ Fall 13 ☑ Spring 14 ☑ Summer 14

Human cytomegalovirus (HCMV) is a medically important herpesvirus and the leading viral cause of congenital birth defects in developed countries. One of its earliest products, the UL37 exon 1 protein or vMIA, is a strong antiapoptotic product. vMIA traffics from the endoplasmic reticulum (ER) to mitochondria through mitochondria associated membrane (MAM) sub-compartment of the ER. Using a combination of genetics, sub-cellular fractionation, confocal microscopy, and super-resolution microscopy, we are determining the mechanisms underlying its trafficking from the ER to mitochondria. In addition, we study how HCMV vMIA inhibits programmed cell death (apoptosis) by increasing the proteasome mediated degradation of proapoptotic Bax and recruitment of the apoptosome to MAM lipid rafts during HCMV infection.

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Dr. Colonnese: Availability: ☑ Fall 13 ☑ Spring 14 ☑ Summer 14

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:**  **Availability:** ☐ Fall 13 ☐ Spring 14  ☑ Summer 14

Genetic and cellular mechanisms underlying neural specification and formation of circuits that regulate emotional and innate behaviors

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**Dr. Crandall:**  
*Availability:* ☑ Fall 13 ☑ Spring 14 ☑ Summer 14

My research program has three main aspects. The first and central component is work on the development and testing through computer simulation of methods for the analysis of DNA sequence data. We have developed methods for estimating gene genealogies, detecting recombination, detecting selection, and measuring genetic diversity and demographic events in the history of a population. We develop software to implement many of these methods and then develop software to test our methods and many others by comparison through computer simulation. Through comparison and tests of robustness to assumption violations, we can gain great insights into why particular methods perform well or poorly and then are in a good position to redevelop improved methodology. In fact, we are currently embarking on the development of a comprehensive simulation software package that allows one, for the first time, to examine the impact of a host of population genetic phenomena at the same time (e.g., migration, mutation, recombination, selection, fluctuating population sizes, tracking geographic locations of alleles in a population, multiple locus populations, etc.). Through such studies, we gain a better understanding of the methodology we use to infer evolutionary and population demographic histories and associated parameter estimates when we apply them to empirical data. Furthermore, such studies provide valuable insights into the development of new and improved theory and methodology for inference from DNA sequence data.

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**Dr. Fu:**  
*Availability:* ☑ Fall 13 ☑ Spring 14 ☑ Summer 14

My lab focuses on breast cancer early detection and management using molecular biology, genomics and bioinformatics tools.

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**Dr. Hathout:**  
*Availability:* ☑ Fall 13 ☑ Spring 14 ☑ Summer 14

Metabolomics and proteomics studies of muscular dystrophies to define novel biomarkers to monitor disease progression and response to treatments.

Define novel therapeutic targets for pediatrics incurable diseases such as Duchenne muscular dystrophy and neurodegenerative disease via study of molecular mechanism of the pathogenesis using cell cultures, mouse models and human clinical samples.

Study of efficacy and risks of novel promising drug for muscular dystrophy.
Techniques: cell cultures, work with animal models, work with human clinical samples, proteomics, metabolomics and genomics methods and learn how to use mass spectrometry and bioinformatics tools.

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**Dr. Hawdon**  
**Availability:** ☑ Fall 13 ☑ Spring 14 ☑ Summer 14

We are developing transgenesis and reverse genetic techniques in parasitic nematodes. We work with animal parasitic nematodes, insect parasitic nematode, and free-living nematodes. We also study the molecular and neurobiology of nematode infection and development.

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**Dr. Hoffman:**  
**Availability:** ☑ Fall 13 ☑ Spring 14 ☑ Summer 14

Research focuses on systems biology of muscular dystrophy, and exercise physiology, as well as drug development and experimental therapeutics.

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**Dr. Jaiswal:**  
**Availability:** ☑ Fall 13 ☑ Spring 14 ☑ Summer 14

Our bodies are constantly exposed to injuries, and injuries have remained a leading cause of death and disability throughout human history. Our lab is interested in understanding the biology of how our body responds to injury and how we can tip this response in favor of repair and regeneration. With the ability to repair such that the repaired tissue can exhibit enhanced function, skeletal muscle is a unique tissue in our body. In an effort to learn about approaches to improve cell and tissue repair we study how muscle cells and tissues repair and regenerate. We employ advanced live cell and tissue imaging approaches together with subcellular and temporal proteomics analysis to discover and study the cell biological and molecular processes leading to efficient repair. An undesired consequence of the remarkable healing ability of skeletal muscle is that genetic defects that interfere with this process lead to debilitating muscular dystrophies and myopathies. We are thus also working towards applying our findings to develop therapeutic approaches for such muscle diseases.
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**Dr. Jeremic:**  
**Availability:**  ❝ Fall 13  ❝ Spring 14  ❝ Summer 14

With aid of modern microscopy, spectroscopy and biochemical methods my laboratory investigates the molecular mechanism of amylin aggregation and amylin-induced oxidative stress in pancreatic beta-cells, hallmarks of type two Diabetes Mellitus (TTDM). Amylin is a thiol-sulfur containing peptide secreted together with insulin from the pancreas and TTDM is understood to be an insulin problem. While the contributions of insufficient insulin release, peripheral insulin resistance, and obesity in the etiology of diabetes are well understood, it is not clear why amylin aggregates in the pancreas and how this aggregation process contributes to the disease. TTDM is chronic metabolic disease that affects over a hundred million people worldwide, and that number is projected to double in two decades. Many hypotheses were proposed over the years to explain the cause for diabetes, but a unifying hypothesis linking diabetic factors such as oxidative stress and pancreatic amyloid is missing. Studies exploring causal connection between amylin aggregation and radicals formation in the pancreas and their roles in the etiology of TTDM are particularly lacking. Understanding how amylin aggregates, and how these aggregates induce oxidative stress in insulin secreting human pancreatic islet b-cells are major topics in the diabetic field, and also represent the main research focus of my laboratory.

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**Dr. Kahn:**  
**Availability:**  ❝ Fall 13  ❝ Spring 14  ❝ Summer 14

My laboratory studies the development of CD8+ T cell immunity in response to intracellular pathogens like Toxoplasma gondii and Encephalitozoon cuniculi. The factors involved in generating robust CD8+ T cell immunity against these infections and strategies involved in their long-term maintenance are being evaluated. These studies will enable to develop therapeutic agents against these agents and information can be extended to other pathogens.

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**Dr. Kusner:**

**Availability:**  ☑ Fall 13  ☑ Spring 14  ☑ Summer 14

The focus of the lab is the neuromuscular junction of skeletal muscle. We are looking at therapeutics that will target the neuromuscular junctions and concentrate a complement inhibitor. We determine the effectiveness of the therapeutic by assessing complement deposition due to a disease model of myasthenia gravis. We are also looking at proteins that localize to the neuromuscular junction in various skeletal muscle types, in particular, the extraocular muscles. We hope to determine whether expression of these protein (or lack of) will influence the susceptibility of skeletal muscle types to specific diseases that influence the function of the neuromuscular junctions.

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**Dr. LaMantia - Dr. Maynard Availability:**  ☑ Fall 13  ☑ Spring 14  ☑ Summer 14

The lab rotation available would be to study the mechanisms by which neural stem cells become specified in the olfactory system.

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**Dr. Lee:**

**Availability:**  ☑ Fall 13  ☑ Spring 14  ☑ Summer 14

**Cancer health disparities:** There are striking population (race) disparities in prostate cancer risk and survival outcome borne out of current health statistics data. This is particularly evident between African Americans (AA) and their Caucasian American (CA) counterparts. Epidemiologic studies have shown that higher mortality and recurrence rates for prostate cancer are still evident in AA men even after adjustment for socioeconomic status, environmental factors and health care access. Thus, it is likely that intrinsic biological differences account for some of the cancer disparities. Our overarching hypothesis is that the biological component of prostate cancer health disparities is due, in part, to population-dependent differential splicing of oncogenes and tumor suppressor genes in cancer specimens. The application of genomic approaches has identified splice variants in AA specimens that are different from CA specimens, and the AA variants appear to encode more aggressive oncogenic proteins, thereby producing a more cancerous phenotype.

The goal of this project is to clone the AA and CA splice variants and to ectopically over-express these variants into tumor cell lines for in vitro and in vivo characterization. Techniques will include, for example, cDNA cloning, tissue culture, western blot analysis, genomics, functional genomics, immunohistochemistry, immunocytochemistry, invasion and proliferation assays, xenografts in mouse models of metastasis.
Dr. Limperopoulos: Availability: ☐ Fall 13 ☑ Spring 14 ☑ Summer 14

My research activities focus on studying the causes and consequences of early life brain injury in high-risk fetal, preterm, and full-term infant populations. Central to my research is the application of advanced magnetic resonance imaging techniques to identify important biomarkers and understand the timing and evolution of brain injury, as well as the brain’s adaptive response following injury. The long-term goals of my research program are to develop reliable biomarkers of brain injury that will guide medical, surgical, and rehabilitation interventions aimed at circumventing injury and minimizing long-term developmental disability.

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Dr. Manzini: Availability: ☐ Fall 13 ☑ Spring 14 ☑ Summer 14

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 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 human disease, understand pathogenesis and study the molecular mechanisms of development. For more details please visit our website www.manzinilab.org

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Dr. Mazumder: Availability: ☐ Fall 13 ☑ Spring 14 ☑ Summer 14

Computational Biochemistry and Molecular Biology strongly rooted in evolutionary biology form the basis of my research program. Many of our predictions have been validated in the laboratory, and there are currently a few that are in the wet-lab testing phase. Our current research goals include conducting a comprehensive comparative analysis at the genomic level (next-generation sequence data) to connect sequence variation to phenotype for cancer genomics studies and better understand Hepatitis C virus infections. We use knowledge derived using bioinformatic approaches in conjunction with experimental data to identify potential experimental targets for development of diagnostics/therapeutics.
Dr. Mendelowitz:  Availability: ☑ Fall 13  ☑ Spring 14  ☑ Summer 14

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 ambiguus 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. Nagaraju:  Availability: ☑ Fall 13  ☑ Spring 14  ☑ Summer 14

My lab research group is involved in using systems biology approaches to study human diseases. Our current research involves: a) defining the mechanisms of damage and dysfunction in autoimmune and genetic diseases using gene and protein expression profiling techniques, b) identifying molecular mechanisms underlying the sex-based differences in immune response and autoimmune diseases such as lupus and arthritis, c) generating novel mouse models and improving existing mouse models to investigate disease pathogenesis, and d) performing basic and preclinical translational studies in autoimmune and genetic muscle diseases using genetic and pharmacological interventions in mouse models of human diseases.

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Dr. Nazarian:  
**Availability:** □ Fall 13 ❌ Spring 14 □ Summer 14

Our lab works on the molecular analysis of pediatric brainstem glioma. Our techniques include proteomics, genomics and transcriptomics. We use in vitro and in vivo methods extensively to for biomarker validation and preclinical trials.

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Dr. Partridge:  
**Availability:** □ Fall 13 ❌ Spring 14 ❌ Summer 14

Investigation of the behavior of myogenic stem cells during growth and regeneration of skeletal muscle

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Dr. Preciado:  
**Availability:** ❌ Fall 13 ❌ Spring 14 ❌ Summer 14

My lab’s current research interests include otitis media (OM), chronic sinusitis and infantile hemangiomas.

OM is the most prevalent disease in the United States, responsible for a significant burden to public health. Its etiology is multifactorial characterized by overproduction of mucous, with increased expression of mucins. Our group has demonstrated that mucin MUC5B is the predominant mucin glycoprotein present in the middle ear of children with chronic OM. We have also started to elucidate the effects of chronic infection and inflammation on mucin genes in vitro. In June of 2012 we obtained our first R01 grant from the NIH/NIDCD focusing on proteomic networks of OM progression in epithelial models. The specific aims of this grant are as follows: First, we aim to use a proteomic and cytokine secretome profiling approach (SILAC) to determine the in vitro effects of NTHi on a middle ear epithelial cell pro-inflammatory response. Secondly, we aim to analyze the effects of key inflammatory mediators identified in Aim 1 on the expression of Muc5b mucin glycoprotein. Finally, we will then aim to intervene at key molecular steps in the process (predictably Cxcl2, and/or NFkB) to halt progression to COM and middle ear Muc5b over-expression.

As a translational component of our OM program, in collaboration with Dr. Benjamin Shapiro from the U of MD, we are studying an innovative method of middle ear clinical drug delivery through an intact tympanic membrane using magnetic push-through nano-technology in a rat model of acute otitis media.
Also, as part of our research program, 3D cell culture systems have been established using primary nasal epithelial cells in order to determine effects of pathologically relevant stimulations submucosal gland formation in vitro. We have recently been awarded a competitive Flight Attendants Medical Research Initiative (FAMRI) Award to study effects of tobacco products on nasal epithelium in vitro and on patient sinonasal secretion mucin content.

Finally, we are jointly funded to perform a prospective, randomized trial has been set up to compare the effectiveness of propranolol vs. corticosteroids for the treatment of infantile hemangiomas. One of the translational basic science aims, is to characterize the proteomic profile of excreted urinary proteins in infants with hemangiomas. Preliminary results demonstrate that patients treated propranolol demonstrate attenuation of excreted matrix metalloproteinase-9 (MMP-9) in their urine over the proliferative phase of the condition when compared to those treated with prednisone.

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**Dr. Rose:**
**Availability:** ☑ Fall 13 ☑ Spring 14 ☑ Summer 14

My lab focuses on the the biology of airway epithelia in chronic inflammatory respiratory tract diseases like cystic fibrosis (CF), chronic rhinosinusitis (CRS) and asthma. We are using the CF and non-CF lung epithelial cell secretomes (generated by a soon to be graduated IBS student!) to develop and test hypotheses as to why CF cells differentially express specific innate immune response proteins and exhibit a protease/antiprotease imbalance, in order to understand how the mutation that causes CF (mutant CFTR) leads to CF lung disease and mucus obstruction in CF airways. We are also investigating how classical and dissociative steroids repress secretory mucin gene expression in the presence of inflammatory mediators that upregulate mucin genes. Lastly, we are developing and using 3-dimensional model systems to study how various triggers activate genes and pathways that lead to an increased number of submucosal glands in the sinus mucosa of patients with CRS. Specific projects are being carried out in collaboration with junior or mid-level faculty. (IBS web site currently being updated).

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**Dr. Sarvazyan:**
**Availability:** ☑ Fall 13 ☑ Spring 14 ☑ Summer 14

My laboratory works on several research projects, as detailed below:
1) ENGINEERING IMMUNOCOMPATIBLE TISSUES. Embryonic stem cells (ESC) provide a unique opportunity to modify the original source of differentiated cells that can allow to subsequently circumvent their rejection by the host. We are testing a novel strategy of diminishing the expression of Major Histocompatibility Complex class I molecules (MHC I) and up-regulating Fas ligand (FasL) expression in order to minimize rejection of ESC-derivatives by the host. The modified ESC can then be differentiated and seeded into decellularized scaffolds from target tissues, followed by testing their immunogenicity in non-autologous hosts. 

*In collaboration with Dr. Zaruhi Karabekian. Funded by the National Science Foundation.*

2) VISUALIZATION OF RFA LESIONS. Radiofrequency ablation (RFA) aims to produce lesions that interrupt reentrant circuits or block the spread of electrical activation from sites of abnormal activity. Today, there are limited means for real-time visualization of cardiac muscle tissue injury during RFA procedures. We found that the fluorescence of endogenous NADH aids the visualization of injured epicardial tissue caused by RFA. This was true for both blood-free and blood-perfused preparations. Gaps between NADH-negative regions revealed unablated tissue, which may promote postablation reentry or provide pathways for the conduction of abnormal electrical activity. We are currently exploring integration of NADH imaging in the design of new RFA catheters.

*In collaboration with Drs. Matthew Kay, Marco Mercader, KC Armstrong, Terry Ransbury and Omar Amirana. Funded by the private investment company Allied Minds.*

3) HETEROGENEITY OF FLOW & REPERFUSION ARRHYTHMIAS. Tachyarrhythmias within the settings of unstable angina can be deadly as they are the common culprits of sudden cardiac death. The broad goal of our studies is to further understand how the dynamic heterogeneity of tissue metabolism that results from unstable angina breeds arrhythmias. We aim to study mechanisms of ischemia and reperfusion arrhythmias from a new perspective: that of connecting local changes in tissue metabolism caused by perturbations in coronary flow to the resulting disturbances in electrical activity. Ultimately, this will provide new insights into possible therapeutic interventions to prevent sudden cardiac death.

*In collaboration with Dr. Matthew Kay. Funded by the National Institutes of Health.*

4) PLASTICIZERS AND CARDIAC FUNCTION. The cardiac effects of plasticizers remain largely understudied. We have recently shown that one of the most commonly used one - DEHP - markedly decreases the conduction velocity, diminishes the amount of connexin-43 and impairs cell adhesion in cardiomyocyte cultures. DEHP treatment has also disrupted expression of a large number of genes, pointing to pathways contributing to an arrhythmogenic phenotype and made cardiac cells increase their dependence on fatty acids for energy production. Our current studies include assessment of in vivo DEHP effects and examining cardiotoxicity of other plasticizers, such as BPA.

*In collaboration with Dr. Nikki Posnack. Funded by the National Institutes of Health.*

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Dr. Shi: Availability: Fall 13  Spring 14  Summer 14

Structure and functional studies of cancer-related proteins and protein complexes.

1. Skp1/Fbxo30/EG5 protein complex.
2. Sialidase, Neu1, and Neu1/Cathepsin A complex
3. Siglec 10/G, and complexed without sialic acids, with 2,3-linked sialic acids and/or 2,6-linked sialic acids.

Techniques involved: Molecular cloning, protein overexpression, protein purification (FPLC), activity assay, protein assay, crystallization.

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Dr. Triplett: Availability: Fall 13  Spring 14  Summer 14

The Triplett lab is interested in the mechanisms by which sensory neurons are organized during development. We use genetic, molecular and electrophysiological techniques to understand this complex and important developmental process.

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Dr. Vanderver: Availability: Fall 13  Spring 14  Summer 14

The Vanderver Lab houses several translational projects:

1) The Myelin Disorders Project is a large national biorepository for patients with leukodystrophies. It collects DNA, serum, plasma and other biologic samples alongside clinical information and neuroimaging. These samples are used for ongoing biomarker samples in several leukodystrophies as well as an active project for gene discovery including next generation sequencing projects. This resource provides the resources for small projects that are applicable to a short term student project.

2) Molecular Mechanisms in Vanishing White Matter disease is a project studying a disorder of eIF2B deficiency and ER stress dysfunction. Using primary human glial cultures, we are assaying ER stress and eIF2B function, as well as the impact of this genetic disorder directly on myelin formation. This is an opportunity to learn challenging human cell culture skills.

3) Molecular Mechanisms in Aicardi Goutieres Syndrome is a project studying disruption in the human innate immune system caused by accumulation of immune stimulatory DNA. Using
mouse models for the disease and human samples, we are analyzing the target brain cell in this devastating neurologic disorder. This project is an interesting application of neuroimmunology, a growing research field.

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**Dr. Wu:** **Availability:** ✗ Fall 13   ✗ Spring 14   ✗ Summer 14

Our research is focused on understanding the neural circuitry mechanisms underlying hearing-related behaviors and cognitive functions. We’re using electrophysiology, imaging, genetics, and pharmacology techniques to investigate the neural circuits for hearing, music and communication, and how auditory information is stored and retrieved in our brain.

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**Dr. Zeichner:** **Availability:** ☑ Fall 13   ✗ Spring 14   ✗ Summer 14

We conduct basic research on HIV and Kaposi’s Sarcoma-associated herpesvirus (KSHV). We are particularly interested in the control of gene expression and the regulation of viral latency and reactivation. We have projects involving new methods to activate HIV from latency, which could potentially ultimately used to attack the latent reservoir of HIV, projects involving the identification and characterization of new KSHV replication pathways trigger by host cell death, and a novel approach to the in vivo identification of highly active immunogens which may be very helpful in HIV vaccine development.

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**Dr. Zheng:** **Availability:** ☑ Fall 13   ✗ Spring 14   ✗ Summer 14

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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