The research focus of my lab is to understand the cellular pathways that control the entry and exit of HIV into a latent state. These studies are directed towards the identification of novel mechanisms that can safely reactivate and eliminate ("shock and kill") or inactivate latent HIV-1 with the purpose of using this knowledge towards HIV-1 eradication strategies. In particular, we are characterizing two classes of latency-reversing agents previously discovered in my lab; benzotriazin derivatives (Bosque et al., Cell Reports, 2017) and Pathogen Recognition Receptor (PRR) agonists (Novis et. al., Retrovirology, 2013). Furthermore, our previous studies suggest that HIV may have evolved to establish a successful latent reservoir (White, PloS Pathogens, 2016). This is a novel hypothesis that has never been explored before and my lab is currently investigating it.

See more at our website: https://smhs.gwu.edu/bosque-pardos-lab/

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Neglected Tropical Diseases, Functional genomics/ genome editing of platyhelminth parasites; Helminth infection induced cancers.

Please see lab website, http://smhs.gwu.edu/brindley-lab/

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**Dr. Bukrinsky:**  
**Availability:** ☑ Fall 17 ☑ Spring 18 ☑ Summer 18

We are studying the effects of HIV infection and HIV protein Nef on cellular cholesterol metabolism. The study relies on biochemical assays, proteomics, fluorescent microscopy, genetics.

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**Dr. Caldovic:**  
**Availability:** ☐ Fall 17 ☑ Spring 18 ☑ Summer 18

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. Chiappinelli:**  
**Availability:** ☐ Fall 17 ☑ Spring 18 ☑ Summer 18

Our lab focuses on the epigenetic regulation of immune signaling in cancer. Epigenetic therapy is particularly appealing as it provides a way to alter gene expression without changing the DNA itself. We study the epigenetic changes in cancer and how epigenetic drugs can reverse these, specifically focusing on noncoding regions of the genome and the tumor cell immune response.

For additional information, visit the link: https://smhs.gwu.edu/chiappinelli-lab/

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**Dr. Corbin:**  
**Availability:** ☐ Fall 17 ☑ Spring 18 ☑ Summer 18

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

http://joshuacorbinlab.com/

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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:      Availability: ☑ Fall 17 ☑ Spring 18 ☑ Summer 18

Bronchiolitis is the #1 cause of hospitalization in US infants, with ~130,000 hospitalizations annually. Small cohort studies (n<210) suggest that 40-50% of infants hospitalized with bronchiolitis will subsequently develop asthma. The greatest challenges for developing primary prevention strategies for this large group of children are the very early identification of modifiable risk factors and the heterogeneity of asthma. The 35th Multicenter Airway Research Collaboration (MARC-35) study is a 17-center prospective cohort study that completed enrollment of 921 hospitalized infants with bronchiolitis in 2014. In this diverse cohort (53% African-American or Hispanic), investigators have collected biospecimens, including nasal swabs at the index hospitalization (median age 3 months). Follow-up data include biannual parent interviews and medical records to age 5 years, with >90% follow-up to date. We are currently extending this largest, most comprehensive severe bronchiolitis cohort in the world by conducting an in-person examination at age 6 years to diagnose and phenotype asthma and by examining nasal airway microRNA and NFκB signaling mediators/outcomes, at both the index hospitalization and at age 6 years. Among these infants with severe bronchiolitis – a natural experiment – we will have a unique opportunity to identify airway microRNAs associated with incident asthma during an important period of lung development that would provide a critical window for primary intervention. Furthermore, using innovative approaches, we will not only investigate underlying mechanisms linking bronchiolitis to incident asthma (e.g., enhanced NFκB signaling) but also identify phenotypes/endotypes of asthma that are likely to respond differently to different interventions. The study will provide a strong evidence base for primary prevention through the future development of targeted interventions (e.g., microRNA-targeting therapy).

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Dr. Gupta: Availability: Fall 17  Spring 18  Summer 18

The Gupta’s lab studies epigenetics in cancer. The long-term goal of my research team is to improve the treatment of cancer patients through the understanding of deregulated oncogenic signaling and epigenetic/genetic regulatory machinery. Despite major therapeutic advances in the cancer treatment, most of the malignancies still remain incurable. New treatment strategies based on improved understanding of the mechanisms of cancer cell growth are needed. Over the past three decades, diagnosis and treatment approach to non-Hodgkin lymphoma (NHL) has been based on the underlying histologic and clinical features, which failed to produce significant advances in the outcome but exposed a large number of patients to potentially toxic therapies. Recent advances in tumor biology have led to the identification of a variety of intracellular oncogenic pathways as potential targets for cancer therapy. Epigenetic and genetic alterations in human lymphoma cells frequently result in deregulation of signal transduction pathways and likely contribute to the multiple oncogenic processes, providing a rationale for pathway–based lymphoma therapy. This emerging concept represents a potential paradigm shift in treatment of lymphoma and other human malignancies, as it advocates basing treatment decisions on the presence of specific deregulated oncogenic signaling pathways irrespective of the histological subtypes. However, for this strategy to be successful, it will be imperative to identify clinical biomarkers that can measure the pathway activation and can be used to match pathway-targeted drugs. Ultimately this approach might help realizing the most cherished goal of personalized medicine to match patients with the most effective cancer therapy in a “personalized manner”.

My laboratory’s research interest fits into the following projects:

1) Oncogenic role of signaling pathways such as JAK/STAT, PI3K/AKT, mTOR, eIF4E responsible for neoplastic transformation and cancer cell growth.
2) To uncover epigenetic regulatory events in cancer (Lymphoma and Melanoma).
3) To discover role of long non-coding RNAs in cancer (Lymphoma and Melanoma).

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Dr. Hashimoto-Torii: Availability: Fall 17  Spring 18  Summer 18

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.

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Dr. Hawdon: Availability: Fall 17 Spring 18 Summer 18

Transgenesis and reverse genetics in parasitic nematode
Molecular biology and immunology of nematode infection
Novel natural products for nematode control

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Dr. Horvath: Availability: Fall 17 Spring 18 Summer 18

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. Hovel-Miner: Availability: Fall 17 Spring 18 Summer 18

Parasite genetics and infectious disease research.

Contact:
The Jones Laboratory is a member of the BELIEVE collaboratory project on HIV cure within the Department of Microbiology, Immunology, and Tropical Medicine at the George Washington University School of Medicine and Health Sciences in Washington, D.C.

Modern therapies can dramatically improve the health of people living with HIV who have access to care, but cannot cure infection. We are committed to harnessing cellular immune responses (T cells and natural killer cells) to improve upon the status quo, by developing therapies that are able to either cure infection, or to further restore health by reducing viral reservoirs.

A variety of rotation projects are available within this overarching theme including: i) studying the impact that T cells and NK cells have on HIV reservoirs from patients samples to gain clues as to how some cells resist elimination; ii) enhancing T-cells and NK cells with nanoparticles to increase their anti-viral activity in vitro and in vivo; iii) studying samples from cohorts of people living with HIV to gain insights into ongoing interactions between the immune system and persistent HIV infection; iv) understanding the role of the central nervous system as a reservoir for HIV, and the potential for T-Cells to eliminate this reservoir.

The primary goal of Dr. Jose’s research is to determine the genetic and pharmacogenetic bases of human essential hypertension and the metabolic syndrome. Specifically, the mission of his laboratory is to study the role of dopamine, adrenergic, and angiotensin receptor subtypes and dopamine regulatory genes (e.g., G protein-coupled receptor kinase 4 [GRK4], sorting nexins, gastrin) on sodium transport in specific nephron segments and their roles in the pathogenesis of genetic hypertension and metabolic syndrome. Dr. Jose’s studies are performed in vitro and in vivo, using molecular, cell biological, and integrative physiological methods, including gene silencing, gene rescue (e.g., AAV vectors), confocal microscopy and biophysical imaging and integrated physiology in rats, mice, and humans. Dr. Jose is a recognized expert on the role of dopamine receptors in the regulation of renal function, epithelial sodium transport, vascular function, and blood pressure. He has contributed novel and important information on signal transduction and cellular trafficking of dopamine receptors. A key finding of Dr. Jose’s research is the demonstration of the crucial role of gene variants of GRK4 in the pathogenesis and personalized treatment of hypertension. These studies have
resulted in two patents in the USA. Deciphering the role of GRK4 gene variants in the pathogenesis of human essential hypertension was the second advance and discovery cited by the Director of National Heart, Lung, and Blood Institute for its 2004 budget justification to the US Congress.

The research in Dr. Jose’s laboratory (nine researchers) is funded by six grants from the National Institutes of Health and one grant from a pharmaceutical company.

1. 7R37HL023081-37 (NIH/NHLBI) - Renal dopamine receptor regulation and function. The major goals are to study the mechanisms of dopamine D1 receptor-, sorting nexin-, and protein phosphatase-regulated renal epithelial cell signaling, renal sodium transport and blood pressure regulation.
2. 7R01DK039308-29 (NIH/NHLBI) - Renal dopamine-1 receptor defect in hypertension. The major goal of this project is to determine how the gastro-renal reflex regulates sodium balance and blood pressure.
3. 2R01HL092196-09 (NIH/NHLBI) - GRK4 and development of salt sensitivity. The major goal of this project is to study the mechanisms by which the GRK4 486V gene promotes salt sensitivity while the GRK4 wild-type gene promotes salt resistance.
4. 5P01HL068686-14 (NIH/NHLBI) - Renal vascular oxidative stress in hypertension. The major goal of subproject #3 is to determine the mechanisms by which the D5 receptor regulates reactive oxygen species.
5. 1R01DK090918-05 (NIH/NIDDK) - Role of dopamine D2 receptors on renal inflammation. The major goal of this project is to determine the role of dopamine D2 receptors in the development of renal inflammation and injury.
6. ZNA35786 (BIAL). Effects of BIA 5-1058 on blood pressure in hypertensive hGRK4 A142V transgenic and D5 receptor knockout mice.

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Dr. LaMantia/Dr. Moody: Availability: ☑ Fall 17 ☑ Spring 18 ☑ Summer 18

The Dysphasia Project is group project under the combined leadership of Dr. LaMantia and Dr. Moody. Students will have the opportunity to learn about bioinformatics via analysis of RNA-seq data, mouse genetics using the 22q deletion mouse model mimicking the various clinical aspects of dysphasia, and cutting edge confocal microscopy with high powered imaging capacity. The rotation project will focus on the role of mitochondrial metabolism in regulating distinct phases of cortical projection genesis and differentiation. This project will provide basic experience in mouse genetics, primary neuronal culture, and molecular methods.

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Dr. Liu: Availability: ☑ Fall 17 ☑ Spring 18 ☑ Summer 18

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. Lynch: Availability: ☑ Fall 17 ☑ Spring 18 ☑ Summer 18

The research focus of the Lynch lab is to study B cell responses to virus infection. Specifically, we are investigating the impact of broadly neutralizing antibodies (bNAbs) on both the HIV-1 virus as well as HIV-infected cells to explore new avenues for the development of potent preventative and therapeutic agents against HIV infection. We have recently demonstrated in human trials that antibody VRC01 can induce escape mutations in the virus of chronically infected individuals (Lynch et al., Science Transl Med., 2015). As human trials with combinations of multiple bNAbs will be assessed in the future, questions related to how the virus escapes from bNAbs must be answered for the design of optimal monoclonal antibody therapy. We are currently mapping virus escape from bNAbs using in vitro replication assays to assess selection pressure of various bNAb combinations on genetically diverse viruses, including non-clade B viruses. We are also analyzing the susceptibility of re-activated reservoir HIV-1 to a panel of bNAbs as well as cloned virus from individuals who cannot control HIV-1 while on anti-retroviral therapy. These studies will inform personalized HIV-1 treatment. We currently study early responses to Zika infection and how these antibody responses may be shaped by prior flavivirus infection. We are analyzing a large cohort of individuals from Colombia to measure their plasma neutralization of Zika virus and Dengue serotype 2.

See more at our website: http://smhs.gwu.edu/lynch-lab/

Contact:
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Dr. Marvar:  
**Availability:** ☐ Fall 17 ☒ Spring 18 ☒ Summer 18

Our laboratory studies the link between stress and anxiety-related disorders (i.e., PTSD) and cardiovascular disease (CVD) risk. We focus on integrative mechanisms related to the brain neurocircuitry involved in the autonomic control of blood pressure and inflammation in hypertension and utilize various animal models to seek clinical and translational therapeutic applications.

Additional information available by consulting the link: https://smhs.gwu.edu/news/common-hypertension-treatment-may-reduce-ptsd-symptoms

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Dr. Miller:  
**Availability:** ☐ Fall 17 ☒ Spring 18 ☒ Summer 18

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 17 ☒ Spring 18 ☒ Summer 18

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 in vivo and in vitro models that we have engineered to develop therapeutics for treating DIPGs.

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**Dr. Penn:**

Neuroplacentology links 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 rodent models of preterm brain injury, we are testing these hormonal neurodevelopmental mechanisms at the cellular, anatomical and behavioral level and correlating our findings in human populations.

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**Dr. Polter:**

Research in my laboratory focuses on the neurobiological effects of stressful and adverse experiences. Our goal is to understand the mechanisms of stress-induced changes in synapses and circuits. We are particularly interested in synaptic regulation of neurons that produce monoamine-neurotransmitters such as dopamine and serotonin that are important modulators of affective and reward-related behavior. Our approach is to use slice electrophysiology coupled with retrograde tracers and viral-mediated optogenics to identify and characterize circuit-specific alterations in synaptic plasticity and function following stress. We then use in vivo manipulations of neural activity to reverse stress-induced neurobiological changes and maladaptive behavioral responses. Our hope is that these studies will provide insight into stress-linked illnesses including depression, PTSD, and substance use disorders.

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**Dr. Posnak:**

Our laboratory investigates the impact of medical devices and procedures on cardiovascular function. Specifically, our recent work has focused on how endocrine disrupting chemicals, which are used in the manufacturing of medical devices and consumer products, can alter cardiac electrical and mechanical function. Our laboratory collaborates on a number of clinically-relevant projects that aim to improve patient outcomes following cardiac surgery and/or transfusion procedures. The laboratory utilizes a wide-array of cardiovascular models (neonatal cardiac cells, human stem cell-derived myocytes, whole hearts, in vivo radiotelemetry), imaging
modalities (confocal, optical mapping, hyperspectral) and phenotypic assays (metabolic, gene expression arrays, calcium and voltage dyes). Our laboratory is a part of the Children’s National Heart Institute and the Sheikh Zayed Institute for Pediatric Surgical Innovation.

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**Dr. Sen:**

I am currently funded by three Am Diabetes Association-Investigator Initiated Studies focusing on the use of CD34+ and CD31+ cell number, function and gene expression in evaluating endothelial dysfunction in patients with early type 2 diabetes, pre- and post-oral incretin mimetic therapy. The fourth project, funded by GW internal research award funds is based on cell metabolism studies 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 adult stem cells as it is (de-novo) as a biomarker in diabetes and also use stem cells post genetic modification using a non-integrating DNA virus such as Adeno or Adeno-associated virus to upregulate or silence a gene of interest in stem cells for regenerative purposes.

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**Dr. Tyagi:**

In our lab, we focus on various aspects of HIV lifecycle:

1) HIV latency, which is 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. In addition to studying the involved pathways, we used this system to identify the biomarkers of latently infected cells, as HIV field still lacks the biomarker that can distinguish latently infected cell from uninfected resting cells.

2) Drugs of abuse, we mainly study the impact of cocaine on HIV gene expression and replication. Pertaining this investigation our main goal is to characterize the molecular and epigenetic pathways that cocaine stimulates in order to influence HIV life cycle.

3) HIV/HCV coinfection and aging process: Since, HIV infected population acquires age related diseases 10 to 20 years earlier than uninfected individuals. Therefore, using patients’ samples we analyze how co-infections such as HCV to HIV patients affect HIV and HCV life cycle and pathways responsible for accelerating aging process in HIV/HCV co-infected patients.
Brain development consists of multiple dynamic processes ranging from cell proliferation, differentiation and migration to neuronal circuit formation and refinement. Each of these processes has vulnerability to various genetic and environmental factors that cause structurally subtle yet functionally serious abnormalities in the brain. The goal of our research is to decipher the complex mechanisms in which these factors impact normal brain development, particularly the cerebral cortex where higher cognitive functions are carried out, and to translate our findings into the development of novel therapeutic approaches for neurodevelopmental disorders such as schizophrenia and autism. Our current focuses include the processes in which various neuronal subtypes are assembled into functional cortical columns and establish specific neuronal connections. Toward this goal, my laboratory uses a combination of cutting edge tools and techniques, including in vivo gene manipulation, induced pluripotent stem (iPS) cells, transgenic animal disease models, proteomic and transcriptomic analyses, and cell encapsulation and transplantation.

The Vilain research group addresses a large and diverse set of research questions centered on Gender-Based Biology and Precision Medicine. Basic Research projects fall under the overarching theme of the laboratory, which aims to identify the genetic and epigenetic factors contributing to sexual development as well as mammalian brain development. Our group has discovered new molecular and cellular mechanisms of sex determination during fetal development, identifying genes as well as mutations affecting gonadal development. We are highly interested in identifying novel genes involved in the sex determination pathway as well as identifying novel regulatory mechanisms to understand the pathogenesis of DSD. We approach many of these questions by using whole exome sequencing (WES), next generation genome sequencing (NGS) as well as RNA sequencing, followed by many in vitro and in vivo validation experiments. In addition to better understanding genetic anomalies leading to DSD, our group is also very interested in identifying and better understanding how factors such as testosterone and estrogen influence and shape the male and female sexually-dimorphic brain, leading to variations in adult sexual behavior. We have elucidated novel mechanisms involved in early brain masculinization and have expanded the understanding of the role that epigenetic factors such as DNA methylation play in this process. Our research projects also include those focused on understanding the genetic and epigenetic contributions leading to the spectrum of human
sexuality, often through our genetic analysis of identical twins that are discordant for sexual orientation; one being gay and the other straight.

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**Dr. Villagra:**
Currently I am Assistant Professor at the School of Medicine and Health Sciences at the George Washington University and member of the recently inaugurated George Washington Cancer Center. My laboratory of Tumor Immunology and epigenetics has two major areas of interest:
1. To understand the cellular and molecular mechanism(s) involved in the induction and establishment of anti-tumor responses and,
2. To translate basic science discoveries into novel molecularly-based immunotherapeutic approaches.

Our basic and translational contributions to cancer research have been highlighted by publications in first tier peer reviewed journals, such as Nature Immunology, Blood, Cancer Research, Immunity and Oncogene, among others. Of particular relevance to the field of Cancer Immunology is our recent finding that specific HDACs regulate the expression of the immunosuppressive PD-L1 and PD-L2 in tumors and immune cells. This effect, in turn, influences the antitumor efficacy of checkpoint blockade with anti-PD1 antibodies.

Our research endeavors have been continuously funded since 2009 by NIH as well as from several grants from Research Foundations and Pharmaceutical companies.

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**Dr. Zheng:**
There are three ongoing research directions in my lab:

1. 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. We are 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.

2. Medulloblastoma (MB) is the most common malignant pediatric brain tumor. Despite its prevalence and importance in pediatric neuro-oncology, the genes and pathways responsible for
its initiation, maintenance, and progression remain poorly understood. Genetically engineered mouse models are an essential tool for uncovering the molecular and cellular basis of human diseases, including cancer, and serve a valuable role as preclinical models for testing targeted therapies. Shh pathway plays an essential role in MBs, mutations in this pathway, including PTC, SUFU, SMO as well as amplification of GLI1 and GLI2, are observed in 25%–30% of human MB cases. In this research project, we propose to establish the PTC-+/+/P53-/- mouse MB model in our lab and use it study the role of a newly identified growth factor NDNF (Neuron-Derived Neurotrophic Factor) in MB initiation, maintenance, and progression.

3. Lung cancer remains the leading cause of cancer deaths worldwide. KRAS is one of the most common oncogenic driver mutations in human lung adenocarcinoma. However, it has been challenging to target KRAS therapeutically. It is becoming evident that, during tumor progression, the tumor cell “seed” co-evolves with the surrounding tumor microenvironment (TME) “soil” and that there is substantial crosstalk between the various cell types which promote tumor growth and development. Thus, improving our understanding of the TME and identifying previously unknown tumor-promoting factors may lead to novel therapies for lung cancer. We recently found the lung alveolar type I (AT1) cells represent a previously undescribed cell type in the TME contributing to lung cancer growth. In this research direction, we are collaborating with Drs. Nemes and Peng to combine genetic and chemical approaches with bioinformatics to characterize the tumor-associated AT1 cells and identify novel tumor-promoting factors expressed by these cells.

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Dr. Zhu, Yan: Availability: ☑ Fall 17 ☑ Spring 18 ☑ Summer 18

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 17 ☑ Spring 18 ☑ Summer 18

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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