The effect of cannabinoids on glioblastoma

About 17,000 primary brain tumors are newly diagnosed each year in the USA. Unfortunately, 60% of them are aggressive Glioblastomas (GBM). Despite the best possible treatments available, the average median survival is only 8-12 months, and there has not been significant improvement over the past two decades. Although radiation therapy and chemotherapy are useful in prolonging the survival, they are not devoid of toxic effects. One of the other alternative modes of cancer therapy that has picked up momentum recently includes use of certain herbal derivatives (synergizers, radiosensitizers or tumor cell killers) that are complementary to appropriate conventional treatments. In this project we will investigate the effects of cannabinoids, alone and in combination with ionizing radiation on glioblastoma cells.

Location: HSC
Student Majors Desired: Biology
Class Preferences: N/A
Important Selection Criteria: Cell culture, microscopy, spectrophotometer plate reading

Investigation of vascular inflammatory disease

Vascular disease is epidemic in our society and is getting worse. Our lab studies the molecular mechanisms of atherosclerosis, restenosis, and angiogenesis. We have several mouse models of vascular disease, and we would teach the URP student how to dissect various tissues from these mice, and then process them for histology and immunohistochemistry. Possibility for more molecular techniques if time/situation permits.

Location: HSC
Student Majors Desired: Biology, Chemistry, Pre-med
Class Preferences: Juniors, Seniors
Important Selection Criteria: Biology is required. Molecular or cell biology, anatomy and physiology would be helpful, but not required. Prior lab experience would also be helpful. Willingness to work with mice.
Antibiotic Resistance Plasmids in the Microbiota

Antibiotic resistance plasmids can be found in the gastrointestinal microbiota (GI), however, the reason they are present in healthy microbiota is unclear. Our laboratory studies how antibiotic resistance plasmids can be carried as part of the commensal Enterococcus faecalis community in the GI tract. Projects include understanding how our model antibiotic resistance plasmid pCF10 can enhance the survival of commensal Enterococci in the GI tract. Determining how pCF10 can make Enterococci more virulent by changing the structure of their biofilms. In collaboration with Dr. Queisser in the Mathematics, we use confocal microscopy to develop computational models to study how biofilm structures influence movement of antibiotics and antibiotic-resistant plasmid transfer events in the GI microbiota.

Location: HSC: Virtual to start, but could shift to in-person if the University approves
Student Majors Desired: Biology, Chemistry
Class Preferences: Freshmen, Sophomores, Juniors, Seniors
Important Selection Criteria: The laboratory accepts students with any level of training and experience.
Hybrid Archaeosomes with Extraordinary Stability and Controlled Release Capability

This research aims to design, construct, and test a new thermosensitive liposome drug delivery system that can overcome the issues of stability, controlled release, and target specificity altogether in the same single formulation and achieve a much higher anticancer efficacy compared to conventional thermosensitive liposomes. Specifically, we plan to develop hybrid archaeosomal drug containing archaeal tetraether lipids and diester lipids, which will be extraordinarily stable and inactive at the body temperature and can be turned to actively deliver drug molecules and kill cancer cells in the tumor areas that are subject to local hyperthermia treatment (i.e., from 37 to 42-44oC). The ultimate goal is to develop this smart material-based liposome formulation into a new clinical approach that will significantly increase the overall anticancer therapeutic efficacy and reduce the side effects of chemotherapy. Through this project, students can learn how to grow thermoacidophilic archa, isolate tetraether lipids from archaea cells, make archaeosomes and archaeosomal drugs, characterize physical properties of archaeosomal drugs and their interactions with serum proteins and mammalian cells.

Location: HSC: Virtual to start, but could shift to in-person if the University approves
Student Majors Desired: Chemistry, Biology, Biophysics
Class Preferences: Freshmen, Sophomores, Juniors, Seniors
Important Selection Criteria: general chemistry

Parkson Chong
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LKSOM
Medical Genetics & Molecular Biochemistry
Effects of In Utero Alcohol Exposure on Biomarkers of Behavioral Dysregulation

There is a high incidence of behavioral dysregulation in children with fetal alcohol spectrum disorder (FASD), but it is not known whether this is due to biochemical changes in the alcohol-exposed fetal brain, or merely reflects the stressful life experiences of children suffering from FASD. This collaborative project will make use of a unique repository of fetal brain-derived exosomes from maternal blood samples, to determine whether the fetuses show abnormalities in the serotonin/dopamine/norepinephrine biochemical pathways, and whether these changes are due to the effects of alcohol or to maternal depression. The results could suggest novel approaches to preventing neuro-psychiatric disorders in children exposed to Alcohol during their fetal development.

This is a proposal to use non-invasive methods to determine whether maternal alcohol use and/or depression affect the synaptic pathways in the fetus that are associated with the behavioral dysregulation of fetal alcohol syndrome (FAS). Children with FAS, or more generally, fetal alcohol spectrum disorder (FASD) exhibit cognitive deficits and affective dysregulation, including irritability, hyperactivity and even depression, which may continue into adulthood. The causes of the affective dysregulation are not known, but it may be due to alcohol-induced molecular abnormalities in the fetal brain. Moreover, many women who use alcohol during pregnancy suffer from comorbid depression or anxiety. Both alcohol use and affective disorders in the mother might influence the fetal development of the very monoaminergic pathways that have been implicated in affective dysregulation, i.e., the serotonergic, dopaminergic and noradrenergic receptors, transporters and downstream pathways. In order to determine whether the brains of Alcohol-exposed fetuses have biochemical abnormalities predictive of affective dysregulation, and if so, whether the abnormalities might be due to Alcohol exposure or maternal depression, we have made use of an exclusively available existing biobank of human blood from pregnant women with detailed maternal psychiatric, substance abuse, and prescription medication history. Our collaborative team will examine neurotoxicity, synaptic markers and biomarkers of affective disorders (protein and mRNA) in male and female fetal B-E isolated from the blood of mothers in four clinical groups: 1) alcohol use; 2) unexposed controls; 3) depression; and 4) depression + alcohol use. In order to isolate the effect of clinical depression from the effects of medication, mothers who used antidepressants are excluded. No new subjects will be recruited. The influence of dose and pattern of alcohol exposure, fetal gender, gestational age, maternal ethnicity, maternal age, BMI, and depression status will be analyzed, using propensity score matching, which will allow inclusion of all 221 samples (150 alcohol-exposed and 71 unexposed controls), while providing sufficient statistical power. This unique translational dataset will provide critically important information about the mechanisms of alcohol-mediated neuronal and synaptic injury during the first and second trimesters, and about molecular targets that might lead to long-term neuropsychiatric consequences of early alcohol exposure,
Effects of In Utero Alcohol Exposure on Biomarkers of Behavioral Dysregulation cont.

i.e., the development of affective dysregulation, including irritability and depression, later in life. The data collected could lead to a better understanding of environmental influences on fetal neurodevelopment, and how this is reflected by biomarkers of affective dysregulation. Ultimately, the resulting knowledge could lead to the development of strategies to prevent psychiatric disorders in children exposed to alcohol during fetal development.

Location: HSC
Student Majors Desired: N/A
Class Preferences: N/A
Important Selection Criteria: High scores, motivation, hard working, a passion for science
Approximately six million adults in the US suffering from heart failure (HF), representing a significant health care burden for the nation. A common feature of HF is excessive extracellular matrix deposition by a specialized and differentiated fibroblast population, known as myofibroblasts, in response to injury of the heart. While myofibroblasts help maintain the structure of the injured heart and prevent heart wall rupture, persistence of myofibroblasts results in excessive fibrosis and cardiac dysfunction. Therefore, identifying molecular mechanisms of myofibroblast differentiation in cardiac fibrosis could yield novel clinical targets to delay or reverse the development of HF. Our lab is studying the mechanisms by which metabolism controls the epigenetic reprogramming of myofibroblasts. We hypothesize that stress and injury alter the metabolism of cardiac fibroblasts and affect how small molecule metabolites mediate changes in epigenetics and chromatin structure to activate the myofibroblast gene program.

**Location:** HSC  
**Student Majors Desired:** N/A  
**Class Preferences:** N/A  
**Important Selection Criteria:** N/A
Calcium regulation of mitochondrial function

The powerful second-messenger, calcium, regulates numerous cellular functions by binding distinct calcium sensing domains or motifs present on numerous proteins. Calcium concentration varies greatly between different cellular compartments, and thus sensors of calcium are strategically localized for subcellular/organelle-specific signaling. Mitochondria actively regulate their calcium concentration and contain calcium sensors to mediate anterograde and retrograde signaling. Mitochondrial Calcium Uptake proteins (MICUs) in the mitochondria contain calcium-binding EF-hand domains, resulting in the regulation of mitochondrial calcium flux, ionic homeostasis, bioenergetics, and cell death. Post-translational modifications and protein turnover are the critical determinants of MICU1 function. We are identifying new regulators associated with mitochondrial calcium flux and their role in pathophysiology using discovery-based approaches, protein biochemistry, high-end imaging-based approaches, and genetic manipulation. We will characterize the pathophysiological consequences of these signaling events in mutant mouse models of disease.

Location: HSC  
Student Majors Desired: N/A  
Class Preferences: N/A  
Important Selection Criteria: N/A
Mitochondrial calcium exchange plays a critical role in regulating cellular bioenergetics, but also contributes to cell death. The overall goal of this project is to understand how alterations in mitochondrial calcium exchange contribute to cardiac injury and heart failure. Our lab has generated numerous genetic mouse models to knock out or overexpress the genes facilitating mitochondrial calcium uptake and efflux in the heart, and we are currently using surgical models of heart failure to assess how perturbation of these pathways protects or predisposes to heart disease. We are also taking in vivo and in vitro approaches to understand the molecular mechanisms that regulate the activity of these calcium handling proteins in order to understand how they could be targeted therapeutically. Students will gain experience in standard cell and molecular biology techniques as well as mouse handling, genotyping, and cardiovascular phenotyping. We seek driven, dependable individuals for this project.

Location: HSC
Student Majors Desired: N/A
Class Preferences: N/A
Important Selection Criteria: N/A
Mitochondrial function in Alzheimer’s disease

Currently, ~5.8 million Americans suffer from Alzheimer’s disease (AD) and there remain no approved therapeutics to lessen the associated neuronal dysfunction and cell death. Various AD clinical trials targeting the “amyloid cascade” have proven unsuccessful, suggesting a dire need to rethink AD disease progression. Alterations in cellular calcium and mitochondrial function are reported as critical molecular contributors to AD pathogenesis. Our lab has previously shown that genetic modulation of either mitochondrial calcium uptake or efflux is a powerful way to limit mitochondrial dysfunction and cell death in the context of cell stress. As causal evidence of impaired mitochondrial calcium exchange in AD, we generated multiple mutant mouse models to modulate mitochondrial calcium exchange and discovered that impaired mitochondrial calcium efflux proceeds neuropathology and memory decline in AD mutant mouses models. In this project, we hypothesize that mitochondrial calcium overload is a primary contributor to AD pathology by promoting metabolic dysfunction and neuronal cell death, and that reducing mito-calcium uptake will impede neurodegeneration and AD pathogenesis. Optimally, these studies will identify new therapeutic targets for the treatment of AD.

Location: HSC
Student Majors Desired: N/A
Class Preferences: N/A
Important Selection Criteria: N/A
Novel genes regulating heart failure

The advent of genomic sequencing revolutionized the field of science, allowing for the annotation of the entire human genome. This sequencing led to the discovery that ~90% of the human genome is transcribed from DNA to RNA, but only ~1% has been identified as being translated into protein. While there are many possible functions of the untranslated RNA, some of these transcripts have the potential to be unidentified protein-coding genes. One reason for this discrepancy in annotations, is that small transcripts are typically excluded from both biological and bioinformatic peptide discovery applications, due to the high rate of misidentification. Recent studies, have identified the translation and biological functions of numerous small transcripts, highlighting the need for re-analysis of this subset of the genome. To discover novel genes involved in the development of heart failure, our group has created a bioinformatic pipeline to identify differentially expressed transcripts with protein coding potential. To date we have predicted coding potential for 26 novel genes and are in the process of confirming translation before determining function.

Location: HSC
Student Majors Desired: N/A
Class Preferences: N/A
Important Selection Criteria: N/A
Novel cell death pathways – focus on necrosis

Necrosis is an umbrella term for the pathological form of inflammatory cell death, common to many chronic diseases. The collective characteristics of necrotic sub-pathways include membrane rupture and the release of cell contents into the extracellular space leading to an immune response. Despite the convergence of these pathways on membrane rupture, the mechanisms underlying the commonalities in necrotic membrane rupture remain unknown. Using a high throughput lentiviral screen, we have identified the SNARE complex as a key regulator of membrane rupture after numerous necrotic stimuli. Our studies have found that knockout of N-ethylmaleimide sensitive factor (Nsf), the protein responsible for SNARE complex recycling, is protective against membrane rupture in the skeletal muscle, heart, and fibroblasts following varying necrotic stimuli. Our ongoing studies delve into the post-translational modifications of Nsf after cell death stimuli. We aim to understand if prevention of Nsf modifications post necrotic stimuli reduces membrane rupture and subsequent cell death. The identification and mechanistic understanding of genes driving cellular necrosis will pave the way for the development of new therapeutics for the treatment of chronic cell death driven diseases.

Location: HSC
Student Majors Desired: N/A
Class Preferences: N/A
Important Selection Criteria: N/A

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LKSOM
Mitochondria-SR Tethering in heart disease

In cardiomyocytes, the sarcoplasmic reticulum (SR) and mitochondria form close interfaces that are maintained by protein tethers which keep the organelles in close apposition. The close proximity between the SR and mitochondria facilitate calcium homeostasis and excitation-contraction coupling which are essential for proper cardiomyocyte function. Abnormalities in SR calcium cycling, transverse-tubule remodeling, and mitochondrial calcium overload are hallmarks of heart failure and contribute to the pathophysiology and progression of heart disease. This project examines the role of the SR protein, PDZD8, in SR-Mitochondria tethering. It employs multidisciplinary approaches ranging from molecular and cell biology, fluorescent, super resolution confocal microscopy and clinically relevant mouse models of heart failure to gain insight into the fundamental principles regarding the role of SR-mitochondria contact sites in cardiovascular diseases.

Location: HSC  
Student Majors Desired: N/A  
Class Preferences: N/A  
Important Selection Criteria: N/A

Genetics and Epigenetics of sex-specific expression patterns in early embryogenesis

We are investigating differences between male and female mouse embryonic stem cells and mouse embryos, and identifying the mechanisms by which these early differences are established. We integrate gene expression, DNA methylation and chromatin conformation analyses with bioinformatics to establish how sex biases affect cellular phenotypes in health and disease.

Location: HSC  
Student Majors Desired: Biology  
Class Preferences: Juniors, Seniors  
Important Selection Criteria: Basic laboratory skills, such as pipetting, running gels, PCR and making solutions required.
Immunometabolism in autoimmunity

Mucosal immunity is highly regulated to sustain the continuous activation by the microbiome and external insults. The immunometabolism of innate immune cells has important consequences on their ability to maintain such regulation and induce immune responses against infections, when necessary. Changes in the main metabolic pathways, i.e. glycolysis, mitochondrial oxidative phosphorylation (OXPHOS) and fatty acid metabolism, dictate whether dendritic cells (DCs) activate or suppress immunity. Modulating the energy metabolism of innate cells can be leveraged for the therapeutic control of immunity. Nonetheless, we have just began dissecting the complexity of the immunometabolism in specialized DC subsets. This project will shed new light into how the innate immune system mounts a response against bacterial infections through the recognition of their most immunogenic components, and it will indicate which immunometabolic pathways can be used as therapeutic targets to fuel responses against antibiotic-resistant chronic infections and to inhibit infection-triggered flares of autoimmunity.

Location: HSC
Student Majors Desired: Biology, Pre-Med
Class Preferences: Sophomores, Juniors
Important Selection Criteria: Hard-working, passionate and able to commit at least 5 interrupted hours in one day.

Deciphering the role of STAT2 in colorectal cancer

Cancer is a very complex disease driven by multiple genetic alterations. The focus of my research is to investigate the mechanism by which the transcription factor STAT2 promotes tumor progression in colorectal cancer. The long-term goal of this project is to determine how STAT2 cooperates with tumor oncogenes to enable tumor progression, conversion of benign lesions to malignant and metastasis. Understanding this process will lead to the development of novel therapeutic interventions to treat colorectal cancer.

Location: HSC: Virtual to start, but could shift to in-person if the University approves
Student Majors Desired: Biology and Biochemistry
Class Preferences: Sophomores, Juniors, Seniors
Important Selection Criteria: No prior research experience is required; Good communication skills, attention to detail and able to follow directions. Self-motivated, eager to learn.
Molecular mechanisms of hypothalamic neurogenesis and neural metabolic syndrome

The research interest in Dr. Hu’s lab focuses on the role and mechanisms of a novel protein NIBP, which regulates NFkB signaling and trans-Golgi networking. Mutation of NIBP contributes to mental retardation, autism, obesity and stroke. In particular, NIBP knockout mice develop obesity under normal diet. Also, the lab is interested in the novel role of the schizophrenia and autism spectrum disorder gene TCF4 in regulating neuritogenesis and synaptic plasticity. The qualified students will actively participate in the daily research activities in the laboratory. These activities include: neural stem cell culture, transfection, reporter gene assay, CRISPR/Cas9 genome editing, molecular cloning, RT-PCR, Western blot, immunohistochemistry, confocal imaging, genotyping, phenotyping and data mining. The students will also participate in the weekly journal club and weekly seminar in the center/department. The students are expected to understand the research publications by Dr. Hu’s group as well as the current progresses in the field of neural metabolic diseases, adult neurogenesis and genome editing. The students with previous research background will be given a small research project that potentially generates publishable data.

Location: HSC: Virtual to start, but could shift to in-person if the University approves
Student Majors Desired: Neuroscience, Biology, Psychiatry, Bioengineering, Computer science
Class Preferences: Sophmores, Juniors, Seniors
Important Selection Criteria: Motivation for science, responsible and reliable
Target-specific delivery of CRISPR/Cas9 genome editors to Disease-relevant cells

CRISPR/Cas9 genome editing has been drawing extensive attention in both science and public. It has revitalized the gene and cell therapy. A large number of exciting and promising preclinical studies escalate the potential of genome editors to treat patients with genetic diseases, infectious diseases, cancer and others. One of many challenges before wide clinical application is the urgent need to effectively, specifically and safely deliver the powerful genome editing machinery to disease-relevant cells and tissues. Dr. Hu’s lab is interested in developing novel viral and non-viral gene delivery for Cas9/sgRNA-expressing vectors or ribonucleoprotein by targeting neural, immune and cancer cells. The qualified students will actively participate in the daily research activities in the laboratory. These activities include: Data mining, molecular cloning, PCR genotyping, real-time PCR, genome editing evaluation, cell culture, transfection, reporter gene assay, Western blot, immunohistochemistry, confocal imaging, etc. The students are expected to understand the research publications by Dr. Hu’s group as well as the current progresses in the field of genome editing and gene/cell therapy. The students with previous research background will be given a small research project that potentially generates publishable data.

Location: HSC: Virtual to start, but could shift to in-person if the University approves
Student Majors Desired: Molecular biology, Genetics, Biology, Neuroscience
Class Preferences: Freshmen, Sophmores, Juniors, Seniors
Important Selection Criteria: High motivation for science, responsible and reliable, hard-working
Development of drug delivery systems with enhanced in vivo stability

Drug delivery systems can modify the pharmacokinetics of drugs, protect them from decomposition and control their spatial and temporal delivery in the organism. In recent years we were involved in the development of drug delivery systems based on amphiphilic compounds of different molecular weight, from simple surfactants, gemini surfactants, lipids, dendrons and polymers. We are currently seeking talented and highly motivated students to develop the next generation of drug delivery systems with enhanced in vivo stability capable of long circulation time in the human body. Students majoring in chemistry, biochemistry and biology are welcomed. Experience in working with cells and animal models is a plus but it is not required.

Location: HSC
Student Majors Desired: Chemistry, Biochemistry, Biology
Class Preferences: Freshmen, Sophmores, Juniors, Seniors
Important Selection Criteria: General knowledge in chemistry, biochemistry, biology and especially in the interdisciplinary integration of this knowledge is needed.

Marc Ilies
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Pharmacy
Pharmaceutical Sciences
Synthesis, physicochemical and biological evaluation of novel carbonic anhydrase inhibitors, activators and their pharmaceutical formulations

Carbonic anhydrases (CAs, E. C. 4.2.1.1) are a class of ubiquitous metalloenzymes that catalyze the reversible hydration of carbon dioxide: \( \text{CO}_2 + \text{H}_2\text{O} \leftrightarrow \text{HCO}_3^- + \text{H}^+ \). Fourteen isozymes are currently known in humans, with different catalytic activity, subcellular localization and tissue distribution. These isozymes are involved in critical physiologic and pathologic processes including respiration, acid-base regulation, electrolyte secretion, bone resorption/calcification, gluconeogenesis, tumorigenicity and the growth and virulence of various pathogens. Some of them are over-expressed in pathological conditions such as edemas, glaucoma, obesity and cancer. Therefore CA isozymes have become important targets for pharmaceutical research. We are seeking talented and passionate individuals to be involved in the synthesis, physicochemical and biological testing of novel selective CA inhibitors and of their pharmaceutical formulations aiming towards treatment of various forms of cancer via novel drugs and drug delivery systems.

**Location:** HSC  
**Student Majors Desired:** Chemistry, Biochemistry, Biology  
**Class Preferences:** Freshmen, Sophomores, Juniors, Seniors  
**Important Selection Criteria:** Previous experience in synthesis, physicochemical and/or biological evaluation of organic compounds and their formulations, as well as towards tissue cell cultures constitutes a plus.
Renal dysfunction after Lung Transplantation

A decline in renal function in the first 6 months after heart or lung transplantation progressively worsens in subsequent years. Furthermore, up to 5% of patients eventually progress to end-stage renal disease (ESRD) requiring permanent renal replacement therapy. The prevalence of renal failure following lung transplant is 26% and 38%, at 1 and 5 years, respectively. However, little is known about the prevalence of RF in the immediate post-operative period following lung transplantation and the long-term consequences of peri-operative RF. This project seeks to determine the prevalence of RF in lung transplant recipients, to identify pre-operative and intra-operative predictive factors for RF, and to assess the effects of RF on long-term renal function and survival.

Location: HSC: Virtual or computational research
Student Majors Desired: Biology, Premed
Class Preferences: Freshmen, Sophmores, Juniors, Seniors
Important Selection Criteria: N/A

Prostate Cancer in Renal Transplant recipients

An increasing number of older patients are being transplanted and as the longevity of the transplanted patients increases, there is an increase in risk of developing prostate cancer. This project aims to determine risk factors for developing prostate cancer in patients with kidney failure and following kidney transplanatation.

Location: HSC: Virtual to start, but could shift to in-person if the University approves
Student Majors Desired: N/A
Class Preferences: Freshmen, Sophmores, Juniors, Seniors
Important Selection Criteria: Data science skills preferred but not an absolute requirement.
Human cardiac progenitor cell exosomes for cardiac regeneration

Injury to the heart leads to development of a scar that never goes away. Current therapies do not target regeneration of the heart of scar replacement. Our current research utilizes cardiac progenitor cells isolated from human heart samples. Tiny vesicles called exosomes from human cardiac progenitor cells that carry immense therapeutic potential will be tested for their ability to repair the heart after myocardial injury.

**Location:** HSC: Virtual to start, but could shift to in-person if the University approves  
**Student Majors Desired:** Biology, Biochemistry  
**Class Preferences:** Sophomores, Juniors, Seniors  
**Important Selection Criteria:** N/A

Neutrophil-endothelial interactions in Sepsis

Sepsis is a major health care problem in the US with more than 1.5 million cases/year and greater than 200,000 deaths/year. This disease is characterized by an intense systemic inflammatory response which activates a cascade of proinflammatory events resulting in tissue damage and organ failure. To date, therapeutic approaches to the treatment of sepsis are largely supportive and there are no specific pharmacologic therapies available. In a translational research project, we are testing the therapeutic effects of a specific kinase inhibitor in a rodent model of sepsis and in an in vitro "tissue on a chip" model. In this project, we will evaluate the effect of this inhibitor on sepsis-induced immune cell migration, programmed cell death (apoptosis), and pathogen clearance. Cell isolation techniques and culture of primary cells and cell lines will be employed. Western blotting, spectophotometric, fluorometric and flow cytometry techniques will be used to determine therapeutic efficacy.

**Location:** HSC  
**Student Majors Desired:** Biochemistry, Chemistry, Biology  
**Class Preferences:** Sophomores, Juniors, Seniors  
**Important Selection Criteria:** Some previous lab experience, highly motivated with an interest in research
The mechanisms of emphysema development

Emphysema belongs to chronic obstructive pulmonary disease and consists of a unique pattern of alveolar wall destruction. There is a very limited effective treatment against this disease. Cigarette smoking induces oxidative stress and is the most common cause of pulmonary emphysema. The goal of this project is to study the mechanism of human alveolar epithelial cell injury and death in this disease development and progression. We validate our results using a murine model of emphysema to further determine the mechanism of alveolar wall destruction. Our obtained results can lead to novel pharmacological strategies to slow the progression of this disease.

Location: HSC: Virtual to start then transition to in-person if University permits
Student Majors Desired: Biology, Biochemistry or related discipline
Class Preferences: Freshman, Sophomore, Junior, Senior
Important Selection Criteria: N/A
CNS regeneration and repair

Our lab is highly interested in neural repair and CNS nerve regeneration research. Our projects focus on the molecular/cellular mechanisms for CNS neuronal growth failure and development of novel and effective strategies to promote neuronal regeneration, remyelination and functional recovery after injury and/or in neurodegenerative disorders. We employ various in vitro and in vivo research approaches, including molecular/cellular neurobiology, biochemistry, genetic and pharmacological methods, transgenic over-expression and knockout mice, and multiple neuronal/axonal lesion models (such as spinal cord injury, optic nerve crush and demyelination of EAE) in mice and rats. We have produced a number of high impact papers related to CNS nerve regeneration and treatments for CNS injury. Our lab is nationally and internationally recognized for discovering that the leukocyte common antigen related phosphatase (LAR) is a receptor for the scare-sourced growth inhibitors of CSPGs and for promoting CNS axon regeneration with available clinical drugs that suppress Rho and GSK-3 signaling pathways. We are currently working on exciting research projects aiming to identify novel genes that regulate CNS neural growth and repair. We currently have ample funds to support graduate study, including two newly-started R01s and similar size of other funds.

Location: HSC: Virtual to start, but could shift to in-person if the University approves
Student Majors Desired: N/A
Class Preferences: N/A
Important Selection Criteria: N/A

Stem cell therapy for cardiac repair

The project would help in understanding different mechanisms that could be involved in heart repair after stem cell or exosomes transplantation after cardiac injury. Immune response is one of the major events that occur after injury. We would study how stem cells can play a part in modulating immune response after myocardial infarction. We will also study interaction of stem cells and other heart cell types including fibroblasts and myocytes.

Location: HSC
Student Majors Desired: N/A
Class Preferences: Sophomores, Juniors, Seniors
Important Selection Criteria: N/A
**Smart Dental Biomaterials for Oral Health**

In our lab, we develop new dental materials for dental applications. Specifically we are interested testing biomaterials that can regenerate oral tissues, and offer antibacterial/anti-fungal effects to prevent infection. We study how these oral microorganisms interact with biomaterials by measuring metabolic activities, biomass, gene expression, etc.

**Location:** HSC  
**Student Majors Desired:** Bioengineering  
**Class Preferences:** Sophomores, Juniors, Seniors  
**Important Selection Criteria:** Lab work, wet lab work, great energy, self-motivation, willing to learn and work in a lab environment, passion for science and new discoveries.

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**Analysis of cerebral vascular changes in a model of traumatic brain injury**

The project entails the use of advanced imaging techniques to determine how capillary networks change following neurotrauma (in a mouse model). This work is important for understanding the recovery of the BBB and vascularization in areas damaged by brain injury.

**Location:** HSC  
**Virtual to start, but could shift to in-person if the University approves**  
**Student Majors Desired:** Any life science  
**Class Preferences:** Sophomores, Juniors, Seniors  
**Important Selection Criteria:** It is preferred that the student has taken some upper level life science classes.
Inflammation in cardiac regeneration and repair

Our research focuses on inflammation and its role in the progression of heart diseases. Inflammation is considered a key element that orchestrate myocardial repair. Although beneficial at early stages, inflammation contributes to myocyte death and subsequent alterations in both the geometry and mechanical properties of the heart. Using different animal models of cardiac diseases (volume/pressure overload or myocardial infarction), we are testing whether inflammatory serine proteases affect cardiac healing, regeneration and function. To better understand the molecular mechanisms involved, we are using a well defined system of cultured cardiac myocytes. Herein, signaling pathways involved in cell survival and death are studied using cellular, molecular, and pharmacological tools.

The main goal of our research is to define new pathways activated by inflammatory proteases and determine if these pathways and serine proteases could be novel targets for therapies that diminish myocyte death and dysfunctional cardiac remodeling after myocardial injury.

Location: HSC
Student Majors Desired: Biology, Biochemistry
Class Preferences: N/A
Important Selection Criteria: Basic knowledge of heart physiology and pathophysiology; Motivated and dedicated students

Aging/AIDS/COVID19

How viruses promote organs to premature and aged

Location: HSC: Virtual to start, but could shift to in-person if the University approves
Student Majors Desired: N/A
Class Preferences: Sophomores, Juniors, Seniors
Important Selection Criteria: N/A
Biochemical basis for HHcy-induced cardiovascular Disease

Our lab studies mechanism underlying hyperhomocysteinemia (HHcy), a medical condition characterized by an abnormally high level of homocysteine in the blood, caused cardiovascular disease (CVD). HHcy is a potent and independent risk factor for CVD, but underlying mechanism is unknown and effective therapy is not available. We are the leading laboratory in this field and the first to report that Hcy selectively activates endothelial cell via hypo-methylation related mechanism and will further explore the biochemical basis of cell type and gene specific methylation in cell and mouse disease models. Each UPR student will be instructed by a PhD student or a postdoctoral fellow.

Location: HSC: Virtual to start, but could shift to in-person if the University approves
Student Majors Desired: Biology, Biochemistry
Class Preferences: Sophmores, Juniors, Seniors
Important Selection Criteria: GPA 3.4 above; 12-15 hours/ week

Metabolic disorder-induced immune cell differentiation

We will identify mechanism mediating cardiovascular inflammation, atherosclerosis and vascular dysfunction. We will characterize immune cell differentiation, vascular and systemic inflammation, vascular cell growth control and apoptosis using bioinformatics, cell biology and molecular biochemical approaches to assess the potential mechanisms mediating metabolic disorder-induced immune cell differentiation. Each UPR student will be instructed by a PhD student or a postdoctoral fellow.

Location: HSC: Virtual to start, but could shift to in-person if the University approves
Student Majors Desired: Biology, Biochemistry
Class Preferences: Sophmores, Juniors, Seniors
Important Selection Criteria: GPA 3.4 above; 12-15 hours/ week
**Vascular energy metabolism (bioenergetics)**

Hyperhomocysteinemia (HHcy) is an independent risk factor for cardiovascular disease (CVD). We will study metabolic cross talk between homocysteine (Hcy) metabolism and glucose metabolism, and analyses glycolysis & mitochondrial respiration in vascular cells. We will use genetic, biology and biochemical tools to characterize the molecular pathway underlying HHcy–altered vascular energy metabolism and its role in CVD. Each UPR student will be instructed by a PhD student or a postdoctoral fellow.

**Location:** HSC: Virtual to start, but could shift to in-person if the University approves  
**Student Majors Desired:** Biology, Biochemistry  
**Class Preferences:** Sophomores, Juniors, Seniors  
**Important Selection Criteria:** GPA 3.4 above; 12-15 hours/week

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**Therapeutic potential of Cannabinoids for Pain and Cognition**

My laboratory currently studies the therapeutic potential of non-psychoactive cannabinoid compounds for the treatment of nervous system disorders, including neuropathic pain, neuroinflammation, cognition, and substance abuse. We use mouse and rat models of these disorders to investigate the safety and effectiveness of constituents of the Cannabis plant, assessing behavioral and molecular outcomes.

**Location:** HSC: Virtual to start, but could shift to in-person if the University approves  
**Student Majors Desired:** Neuroscience, Biology  
**Class Preferences:** N/A  
**Important Selection Criteria:** N/A
**Exploring the relationship between allergy and cancer**

Despite a well-established negative correlation between allergy and cancer risk based upon epidemiological studies, rigorous scientific testing of the functional relationship between allergic inflammation and carcinogenesis represent a significant knowledge gap. Our lab has combined mouse models of esophageal allergy and cancer to demonstrate that exposure to allergic inflammation limits tumor formation and progression in the esophagus. The current project uses an ex vivo co-culture system in which immune cells from mice with esophageal allergy are cultured in the presence of tumor cells isolated from esophagi of mice with esophageal cancer. The goal is to define the immune cell type(s) induced in mice with esophageal allergy that mediate killing of esophageal tumor cells. This represents a novel ideation of cancer immunotherapy with great potential for substantive clinical and translational impact in esophageal cancer patients.

**Location:** HSC  
**Student Majors Desired:** N/A  
**Class Preferences:** N/A  
**Important Selection Criteria:** N/A

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**Identification of novel therapeutic approaches to treat metabolic disorders**

Our current research interests are 1) long non-coding RNAs (lncRNAs) and protein-coding genes in metabolic disorders; 2) RNA or RNA targeted therapies to treat metabolic disorders; and 3) Multi-Omics approach to dissect the pathological process of metabolic disorders. Students will get exposure to both bioinformatics and experimental biology.

**Location:** HSC: Virtual to start, but could shift to in-person if the University approves  
**Student Majors Desired:** Biology, Biochemistry, Computer Science, Mathematics or related majors  
**Class Preferences:** Sophomores, Juniors, Seniors  
**Important Selection Criteria:** Interested in metabolic diseases. Self-motivated and detail-oriented. Knowledge of general molecular biology techniques.
Targeting cardiac fibrosis

Chronic ischemia induced tissue fibrosis contributes to numerous end-stage diseases. Moreover, endomyocardial biopsy specimens from patients with atherosclerotic coronary disease-induced ischemic cardiomyopathy demonstrated 45% of replacement fibrosis. Thus, identifying the signaling cascades that regulates fibrosis in chronic ischemic diseases will have significant clinical benefit. In this project we will use a newly established chronic ischemia-induced cardiomyopathy model to: (1) interrogate the role of a novel signaling pathway that regulates ischemia-induced tissue fibrosis, (2) uncover the molecular mechanisms of its regulation of pro- and antifibrogenic signaling cascade, and (3) test the therapeutic potential of a newly developed allosteric inhibitor against fibrosis in vivo. The success of proposed study will define a drug target-able pathway in regulating chronic ischemia-induced tissue fibrosis. The successful candidate, who has previous lab experience, will have chance to conduct a part of the project and contribute to research article.

Location: HSC
Student Majors Desired: Biology, Biochemistry, or pharmacology
Class Preferences: Sophomores, Juniors, Seniors
Important Selection Criteria: Highly motivated and responsible. Basic cell and molecular biology techniques and understanding of human physiology are a plus.

Molecular control of vascular remodeling and lymphangiogenesis

One of our lab's research focus is to identify novel signaling pathways that regulate vascular angiogenesis and lymphangiogenesis, one of the major cardiovascular problems. Reticulon family proteins (RTN) are mainly localized to endoplasmic reticulum (ER) and regulate mitochondria associated ER membrane (MAM). In this project we are using genetic modified animal models, cellular and molecular techniques to uncover how Nogo-B, the only RTN-4 family protein expressed in vessel wall, regulate MAM remodeling in endothelial cells, and vascular and lymphatic angiogenesis in diabetes.

Location: HSC
Student Majors Desired: Biology, Biochemistry, or pharmacology
Class Preferences: Sophomores, Juniors, Seniors
Important Selection Criteria: Highly motivated and responsible. Basic cell and molecular biology techniques and understanding of human physiology are a plus.