Potential Mentors for MD/PhD students

Wayne State University
School of Medicine

Faculty Profiles
Winter 2023
Elizabeth Berger, Ph.D.

The studies carried out by our laboratory predominately focus on disease pathogenesis and the interplay between the immune and neuroendocrine systems. We mechanistically and therapeutically investigate the events of inflammation and innate immunity using models of ocular infectious disease and diabetic retinopathy. This includes analyses of: pro-resolving lipid mediators, host inflammatory cells (macrophages, neutrophils, T cells - both in vivo and in vitro), extracellular matrix and adhesion molecules, cytokines/chemokines, Toll-like receptors and other related molecules using a number of molecular, cellular and immunohistochemical techniques.

Elizabeth Berger, Ph.D.
Department of Ophthalmology, Visual and Anatomical Sciences Wayne State University eberger@med.wayne.edu

Bruce Berkowitz, Ph.D.

Dr. Berkowitz’s research is focused on developing and applying novel imaging methods to measure neuronal energy ecosystem biomarkers in vivo (including mitochondrial respiration and oxidative stress) to improve treatment efficacy during emerging neurodegeneration.

http://berkowitzlab.wayne.edu/
Google Scholar Link

Bruce Berkowitz, Ph.D.
Department of Ophthalmology, Visual and Anatomical Sciences Wayne State University baberko@med.wayne.edu

Maria Bykhovskaia, Ph.D.

Our research is focused on molecular machinery that controls the release of neuronal transmitters from nerve terminals. Neuronal transmitters are packaged into synaptic vesicles and released by fusion of the vesicles with the neuronal membrane. These processes are highly dynamic and plastic, and disruptions in their regulation can produce severe neurological disorders, such as epilepsy. Our lab investigates the mechanisms that lead from disruptions in the synaptic vesicle fusion to imbalance in neuronal networks to epilepsy.

Maria Bykhovskaia, Ph.D.
Department of Neurology Wayne State University mbykhovs@med.wayne.edu
Our research is focused on the host immune response to corneal infection, in particular, to the gram-negative bacterium Pseudomonas aeruginosa (PA). The first tests whether glycyrrhizin (GLY), a derivative of the licorice root, in combination with antibiotics potentiates antibiotic effectiveness to multi-drug resistant isolates of PA and the mechanisms involved. A second project tests the effects on the eye of exposure to airborne particulates vs ambient air (using a whole-body exposure chamber) and response to bacterial infection of the cornea. The role of the Nrf2 pathway is being tested as well as how to inhibit its signaling and decrease disease. In vivo and tissue culture models are used.

Both healthy aging individuals as well as military personnel that are exposed to loud noise or explosions often suffer from untreatable deterioration in auditory and vestibular function. Studies in the Holt Lab focus on the effect of loud noise and blast on central auditory and vestibular pathways and the impact on function (hearing and balance). To understand the biological basis of dysfunction in these pathways, we collect behavioral, neuronal, molecular, and biochemical, data related to ion channels and neurotransmission. The goal is to both understand the causes of auditory and vestibular dysfunction and identify new targets for treatment and prevention.
Ahmed Ibrahim, Ph.D.

My research centers on investigating the underlying molecular and cellular mechanisms of Angiogenesis-driven diseases. The focus is primarily on pathological ocular angiogenesis which is the underlying mechanism of a variety of sight-threatening diseases such as Retinopathy of prematurity (ROP), proliferative diabetic retinopathy (PDR), and exudative age-related macular degeneration (wet AMD). The long-term goal of my research is to identify novel therapeutic targets, with specific focus on endothelial metabolic reprogramming, and to discover already FDA-approved drug(s) that could be repurposed to modulate the key proteins in the energy metabolism of angiogenic endothelial cells.

Ahmed Ibrahim, Ph.D.
Department of Ophthalmology,
Visual and Anatomical Sciences
Wayne State University
ahmed.ibrahim@wayne.edu

Tomomi Ichinose, M.D., Ph.D.

Our research is focusing on understanding the visual system. Using the mouse and human retina as the model system, we conduct electro-physiology and immunohistochemistry to investigate neural network and visual signal processing. We investigate how retinal neural network processes motion detection, light adaptation, and other visual functions.

Tomomi Ichinose, M.D., Ph.D.
Department of Ophthalmology,
Visual and Anatomical Sciences
Wayne State University
tichinos@med.wayne.edu

Ryan Insolera, PhD

With their high energetic demands, neurons are critically reliant on a healthy population of mitochondria, which are constantly maintained through biogenesis, dynamics, and degradation. In the lab, we are interested in understanding the underlying cell biology of neurodegenerative diseases in which mitochondrial maintenance is disrupted. We primarily utilize the simple model organism Drosophila melanogaster (fruit flies) to achieve the goal of correlating subcellular perturbations with physiological dysfunction and neurodegeneration.

Ryan Insolera, PhD
Department of Ophthalmology,
Visual and Anatomical Sciences
Wayne State University
rinsolera@wayne.edu
Renu Kowluru, Ph.D.

Our main goal is to understand the molecular mechanism(s) of the development of diabetic retinopathy, and research focus is on mitochondrial damage and epigenetic modifications. The lab uses both in vivo (rodents) and in vitro (primary cells in culture) models of diabetic retinopathy, and utilizes molecular, biochemical and functional techniques.

Ashok Kumar, Ph.D.

The research interests of laboratory are to study host-pathogen interactions in microbial infection caused by bacteria, viruses, and fungi. Specifically, our focus has been on developing immunomodulation strategies targeting Toll-like receptor (TLR) signaling in preventing and/or treating infectious diseases affecting the eye. We use cutting-edge and high throughput “omics” technologies such as transcriptomics, lipidomics, and metabolomics combined with systems biology to study host immune responses to infection. Another area of our research is to understand mechanisms of antibiotic resistance among bacteria, and develop alternative antimicrobial therapeutics to reduce antibiotic resistance.

Zhuo-Hua Pan, Ph.D.

Research in my laboratory focuses on the development of optogenetic strategies to cure blindness caused by retinal degenerative diseases. The optogenetic approach involves converting light-insensitive inner retinal neurons to photosensitive cells by ectopic expression of microbial channelrhodopsin (ChRs), thus imparting light sensitivity. Ongoing research projects include: 1) developing low light-sensitive and long wavelength-sensitive ChRs; 2) improving AAV-mediated retinal cell targeting; 3) investigating the impact of retinal degeneration on the outcome of optogenetic vision restoration. Molecular engineering, immunohistochemical analyses, electrophysiological recordings, and animal behavioral tests are performed in our studies.
Vascular diseases are the leading cause of morbidity and mortality in developed societies. My research focuses on understanding the molecular mechanisms of vascular diseases particularly atherosclerosis, restenosis, and proliferative retinopathies. Our multidisciplinary approach includes biochemical and cell biology procedures (Western blot analysis, 2-D gel electrophoresis, HPLC, cell migration assay, transfections, imaging, and immunoprecipitation), molecular techniques (electrophoretic mobility shift assay, chromatin IP assay, site-directed mutagenesis, and cloning), and various animal models (Rat/mouse carotid artery injury, hind-limb ischemia, oxygen-induced retinopathy, and ApoE-/- mouse model). By defining the molecular bases for these pathologies, we hope to find new targets for therapeutics to improve vascular disease prevention and treatment.

My research is on diabetic retinopathy with a focus on retinal inflammation. We use cell culture and animal models to investigate how specific cAMP pathways may protect the diabetic retina through reducing inflammation.

Tissue injury results in an initiation of inflammation. Acute mucosal inflammation developed in response to an infection is beneficial to host, as it helps in eradicating the infectious pathogen. However, the inflammation that persists (chronic inflammation) in mucosal tissue for longer time-period is detrimental to host because it prevents an effective healing and the normal functioning of the tissue. Depending upon the tissue that gets inflamed, chronic inflammation could cause morbidity or mortality to the host. The goal of our lab is to understand the cellular and molecular pathways involved in regulating sterile inflammation and viral infection induced chronic inflammation.
Ryan Thummel, Ph.D.

The main focus of the lab is to study the development and function of the retina. We utilize all the genetic and developmental advantages of zebrafish as a model system – most notably their conservation to our human anatomy and physiology in regard to pigmentation, myelin formation, and visual function. In addition, we take advantage of the amazing capacity of the zebrafish to regenerate its entire retina following injury or disease. [https://www.thummellab.com/](https://www.thummellab.com/)

In addition to his research, Dr. Thummel is the co-course director for the M1 and M2 research elective courses, is a core member for the M1 histology and embryology disciplines, assists with M1/M2 problem-based learning exercises, and assists in teaching the M2 pathobiology laboratory sessions.

Fu-Shin Yu, Ph.D.

We have two NIH/NEI funded projects: corneal innate immunity and diabetic wound healing and sensory nerve (de)regeneration. Using mouse model of diabetes, we investigate how hyperglycemia causes sensory neuropathy and delayed wound healing with focus on the role of exosomes, a newly rediscovered mediator of cell-cell communication. Our second project addresses molecular mechanisms underlying susceptibility and proneness of diabetic patients to microbial infection in the cornea.

Shunbin Xu, Ph.D.

The research interest of my laboratory is to study the roles of microRNAs (miRNAs) in the eye and ocular diseases. miRNAs are small, non-coding, regulatory RNAs and constitute a newly recognized level of gene expression regulation. Our long-term goal is to uncover the roles miRNAs in normal development and function of the eye, as well as in ocular diseases so as to identify novel miRNA-based therapeutic targets for the treatment of various ocular diseases. One of the major projects is to study the roles of miR-183/96/182 cluster in retina and other sensory organs. The second major project in my laboratory is on miRNAs in diabetic retinopathy (DR).
MICROBIOLOGY, IMMUNOLOGY, & BIOCHEMISTRY
Primary Faculty

**Yuan He, PhD**  
Dept. of Biochemistry, Microbiology and Immunology  
Wayne State University  
yhe@med.wayne.edu

Our lab works on the sensing mechanisms of innate immunity. We currently focus on the NLRP3 inflammasome, one of the critical innate sensors that mediate host immune responses to diverse microbial infections and cellular damage. However, aberrant activation of the NLRP3 inflammasome has been linked to the pathogenesis of several inflammatory disorders, including cryopyrin-associated periodic syndromes (CAPS), Alzheimer’s disease, diabetes, and atherosclerosis. Our immediate research goal is to understand the function and regulatory mechanism of NLRP3 inflammasome activation during immune responses. Our long-term goal is to elucidate the function and regulation of inflammasome-forming receptors in innate immunity and to translate those findings into novel treatments for inflammatory diseases.

**Eric Sebzda, PhD**  
Dept. of Biochemistry, Microbiology, and Immunology  
Wayne State University  
eric.sebzda@wayne.edu

The Sebzda lab studies novel aspects of peripheral tolerance as it relates to cancer and autoimmunity. To do this, we utilize unique gene-modified mouse models to understand the basic mechanisms responsible for non-redundant forms of immune tolerance. This has led to ground-breaking discoveries, including the generation of mice that are resistant to cancer malignancy and animal models that replicate the pathology of autoimmune patients. We are currently seeking motivated trainees who wish to join this research venture and obtain first-author publications.

**Jeffery Withey, PhD**  
Dept. of Biochemistry, Microbiology, and Immunology  
Wayne State University  
jwitheym@med.wayne.edu

The Withey lab is focused on enteric bacterial pathogens, with an emphasis on Vibrio cholerae, the cause of human cholera. We developed a zebrafish model for cholera that allows us to study the entire V. cholerae life cycle, both as an intestinal pathogen and as a component of aquatic ecosystems. We are investigating the V. cholerae genes important for intestinal colonization and disease transmission, as well as the fish immune responses to infection and the protective role of the intestinal microbiota. These findings are then translated to mammalian models to identify factors in human disease.
Zhe Yang, PhD  
Dept. of Biochemistry, Microbiology, and Immunology  
Wayne State University  
zyang@med.wayne.edu

Our research focuses on a special class of protein lysine methyltransferases with the evolutionary history dated back to at least 1.5 billion years ago at the beginning of eukaryotic life. The main goal of our research is to uncover new paradigms in these proteins while further broadening our understanding of their functional diversity in both normal biology and disease states, ranging from tumor cell proliferation, cancer stemness and dormancy, the immune response, and cardiomyocyte differentiation.

Not primary faculty

Ashok Kumar, PhD  
Department of Ophthalmology, Visual and Anatomical Sciences; Dept. of Biochemistry, Microbiology, and Immunology  
Wayne State University  
akuma@med.wayne.edu

The research interests of laboratory are to study host-pathogen interactions in microbial infection caused by bacteria, viruses, and fungi. Specifically, our focus has been on developing immunomodulation strategies targeting Toll-like receptor (TLR) signaling in preventing and/or treating infectious diseases affecting the eye. We use cutting-edge and high throughput “omics” technologies such as transcriptomics, lipidomics, and metabolomics combined with systems biology to study host immune responses to infection. Another area of our research is to understand mechanisms of antibiotic resistance among bacteria and develop alternative antimicrobial therapeutics to reduce antibiotic resistance.

Qing-Sheng Mi, MD, PhD  
Dept of Oncology; Dept of Biochemistry, Microbiology and Immunology Wayne State University; qing-sheng.mi@wayne.edu  
Dept of Dermatology, Henry Ford Health; qmi1@hfhs.org

The long-term goals of my research are to uncover the molecular and cellular mechanisms underlying skin inflammatory/autoimmune diseases and cancer progression/metastasis, to identify therapeutic targets and biomarkers related to early diagnosis and therapeutic response. My current NIH funded projects include 1) identification of early diagnostic biomarkers for psoriatic arthritis; 2) Functional immunogenetics of hidradenitis suppurative; 3) TGFb pathways and skin Langerhans cells; 4) Defining the new subsets of skin Langerhans cells. We also study the genetic and epigenetic factors that regulate skin/liver/pancreatic cancer development and serve as immunotherapy targets.
Kezhong Zhang, PhD
CMMG; Dept. of Biochemistry, Microbiology, and Immunology
Wayne State University
kzhang@med.wayne.edu

Research in the Zhang Lab is focused on molecular mechanisms and physiological roles of cellular stress signaling from the endoplasmic reticulum (ER) or mitochondria in inflammation and metabolism associated with metabolic disease, autoimmune disease, and cancer. Specific research projects open to graduate or MD students: Functional significance and mechanistic basis of stress responses associated with dysregulated circadian rhythm, air pollution, and over-nutrition in inflammatory, metabolic, and degenerative diseases, including fatty liver disease, diabetes, and Alzheimer’s Disease.
CANCER BIOLOGY
Asfar Azmi, Ph.D.
Department of Oncology
Wayne State University
azmia@karmanos.org

Dr. Azmi’s lab is focused on translational cancer research. His research group has made fundamental discoveries on the role of aberrant nuclear protein transport in cancer. The main goals of his lab are development of novel therapeutic targets, pre-clinical and early phase development of new drugs for pancreatic cancer and pancreatic neuroendocrine tumors particularly, nuclear export inhibitor selinexor and KRAS pathway targeted therapies. Work done by his team led to the FDA approval of selinexor in several tumor indications. A number of additional early phase clinical trials have emerged from his work and his team is conducting reverse translation studies on these trials.

Jennifer Beebe-Dimmer, Ph.D., MPH
Professor
Department of Oncology
Wayne State University
dimmerj@karmanos.org

Dr. Beebe-Dimmer is an epidemiologist with a research program broadly addressing the determinants of cancer health disparities in populations residing within Karmanos Cancer Institute’s catchment area. She is leader of the Population Studies and Disparities Research Program and Scientific Director of the Epidemiology Research Core. She has several funded grants examining the role of inherited susceptibility to early onset and hereditary prostate cancer in African American men. She is Co-PI of the Detroit Research on Cancer Survivorship which studies the multiplex causes of poorer outcomes in African American cancer survivors. She is also the PI of the CAPABLE study, a 12-week HIIT intervention for cancer survivors.

Frank Cackowski, MD, Ph.D.
Assistant Professor
Department of Oncology
Wayne State University
cackowskif@karmanos.org

Tumors are heterogenous mixtures of billions of cells, and can change their characteristics over time and by location to adapt to their environment. This includes how some cancer cells adopt characteristics of tissue stem cells, which is important for them to initiate new tumors and survive our attempts at treatment. Sometimes this tumor plasticity extends so far as to allow the tumor to completely change its appearance. For example, in prostate and some other cancers, the cells can change from their typical appearance as abnormal duct lining cells (adenocarcinoma) to neuroendocrine cells that appear as if they had originated from completely different part of the body. Similarly, and possibly as an adaptation mechanism, prostate cancer cells can spread
from the primary tumor to distant sites, lay dormant, and then grow into fatal disease years or decades later. These dormant disseminated tumor cells spend much of their time in the resting, or G0 phase of the cell cycle. Analogously, some studies have also shown that most of the cancer stem-like cells are contained within the quiescent, or G0 population. My lab is focused on these overlapping concepts of plasticity, dormancy, and quiescence. We use patient samples, cell culture, animal models, and bioinformatics to understand this biology and develop ways to prevent relapse, deepen treatment responses, and combat treatment resistance.

HyeonJoo Cheon, Ph.D.
Assistant Professor
Department of Oncology
Wayne State University
cheonh@wayne.edu

The Cheon Lab is interested in understanding type-I interferon (IFN-I) responses, which impact the effectiveness of radiation therapy, chemotherapy, and immunotherapy. We Our current research is focused on understanding the immune-independent, cancer cell-intrinsic function of programmed cell death ligand 1 (PD-L1), which enhances the resistance to DNA damage by regulating IFN-I responses. Our research will contribute to innovative approaches to treat therapy-resistant tumors using knowledge of IFN-I synthesis and signaling in cancer cells, which has not been previously understood because of the complexity of IFN biology.

Sreenivasa R. Chinni, Ph.D.
Associate Professor
Department of Urology, Pathology and Oncology
Wayne State University
schinni@med.wayne.edu

The research focus of my laboratory is to understand the molecular mechanism of prostate cancer metastasis and resistant mechanisms to anti-androgen/AR therapies. Currently, we are studying the role of cross-talk between chemokine receptors and phosphatidylinositol 4 kinase IIIα in tumor cells. We recently found that chemokine receptors interact with PI4KIIIα in cancer cells, and this novel interaction contributes to cell invasion. Current work is in progress to critically address how PI4KIIIα is activated in cancer cells and assess the role of evolutionarily conserved adaptor proteins in PI4KIIIα activation within cancer cells. Using cellular and xenograft models of bone metastasis, the role of PI4KIIIα will be determined in intraosseous tumor growth using genetic and pharmacological approaches.

Other projects in the lab are to understand the altered androgen synthesis during castration-resistant disease progression. How transcriptional regulation of androgen biosynthetic enzymes contributes to intracellular production of androgens and promote bone tumor growth. In addition, we have recently collaborated with oncologists to procure metastatic biopsy specimens from prostate cancer patients undergoing anti-androgen/AR therapies, we are also currently
characterizing the altered gene expression profiles during therapies and plan to characterize the therapy-resistant mechanism further using cell and xenograft models.

Yubin Ge, Ph.D.
Department of Oncology
Wayne State University
gey@karmanos.org

Research in the Ge laboratory spans the basic biology of acute myeloid leukemia (AML) in children and adults to translational studies with primary patient specimens and cell line- and patient-derived xenograft mouse models. Ge laboratory studies are currently focusing on the antileukemic activity and molecular mechanisms for novel targeted agents, including histone deacetylase (HDAC) inhibitors (HDACIs), Bcl-2 inhibitors, FLT3 inhibitors, and mitochondrial targeting agents, either alone or in combination, in preclinical models of AML, and novel strategies to combat therapy-resistant AML.

Heather Gibson, Ph.D.
Assistant Professor
Department of Oncology
Wayne State University
gibsonh@karmanos.org

Cancer immunotherapy breakthroughs have shown tremendous promise, but there remains an urgency to determine which patients will respond and why many do not. My lab uses functional genomics and genetically diverse animal models to pinpoint genes and/or pathways involved in immunotherapy resistance. We have discovered putative regulators of both immune checkpoint inhibitors and anti-tumor antibody, also known as targeted immunotherapy. The goal is to identify both biomarkers and actionable targets to improve clinical outcomes. Additionally, we have multiple ongoing collaborative projects aimed at developing new strategies to engage the immune system against cancer.

Benjamin Kidder, Ph.D.
Assistant Professor
Department of Oncology
Wayne State University
benjamin.kidder@wayne.edu

Our lab focuses on cancer and stem cell epigenetics, transcriptional networks, and reprogramming. We are particularly interested in understanding how epigenetic landscapes are regulated in cancer vs. normal cells, the role for heterochromatin in regulating genome stability, and how histone modifying enzymes contribute to the diverse cellular repertoire that exists in mammals.
Jing Li, Ph.D.
Professor
Department of Oncology
Wayne State University
LiJing@wayne.edu

The central theme of Dr. Li's research is to promote rational cancer therapy and drug development by better understanding the clinical pharmacology of anticancer agents. Her research particularly focuses on the mechanistic understanding and quantitative prediction of drug penetration across the blood-brain barrier into the human brain and brain tumors, by employing an integrated translational approach that leverages preclinical pharmacology studies, physiologically based pharmacokinetic modeling, and clinical trials.

Karin List, Ph.D.
Departments of Pharmacology and Oncology
Wayne State University School of Medicine
klist@med.wayne.edu

We aim to understand the physiological role of extracellular proteases in tissue development and homeostasis is important in order to pinpoint how dysregulated proteolysis can cause or contribute to cancer progression. The motivation behind parallel investigations of normal physiology and pathology is the idea that carcinogenesis often involves pathways, including proteolytic pathways, that are important in normal development and have gone awry in cancer. Generation and characterization of mouse models, including models of human cancer, play an integral role in our research. We use knock-out and transgenic mice for selected extracellular proteases and protease inhibitors as unique tools to identify critical proteolytic pathways in health and disease.

Larry Matherly, Ph.D.
Professor
Department of Oncology and Pharmacology
Wayne State University
matherly@karmanos.org

Metabolic reprogramming is a hallmark of cancer. Of the altered metabolic pathways in cancer, one-carbon (C1) metabolism is notable. C1 metabolism encompasses folate-mediated C1 transfer reactions and related processes, including nucleotide and amino acid biosynthesis, anti-oxidant regeneration, and epigenetic regulation. Uptake of folates into tissues is mediated by the major facilitative transporters, the reduced folate carrier (RFC) and the proton-coupled folate transporter (PCFT), and by folate receptors (FRs) α and β. C1 pathways are compartmentalized in the cytosol, mitochondria and nucleus. Current studies in the Matherly laboratory focus on understanding the biology of C1 metabolism and related processes in relation to therapy of cancer, as well as other diseases.
Qing-Sheng Mi, MD, Ph.D.
Professor
Dept of Biochemistry, Microbiology and Immunology; Dept of Oncology
Wayne State University; qing-sheng.mi@wayne.edu
Dept of Dermatology, Henry Ford Health; qmi1@hfhs.org

The long-term goals of my research are to uncover the molecular and cellular mechanisms underlying skin inflammatory/autoimmune diseases and cancer progression/metastasis, to identify therapeutic targets and biomarkers related to early diagnosis and therapeutic response. My current NIH funded projects include 1) identification of early diagnostic biomarkers for psoriatic arthritis; 2) Functional immunogenetics of hidradenitis suppurativa; 3) TGFb pathways and skin Langerhans cells; 4) Defining the new subsets of skin Langerhans cells. We also study the genetic and epigenetic factors that regulate skin/liver/pancreatic cancer development and serve as immunotherapy targets.

Izabela Podgorski, Ph.D.
Professor
Department of Pharmacology & Oncology
Wayne State University
ipodgors@med.wayne.edu

Our main research objectives are to identify molecular mechanisms underlying the association between bone marrow adiposity and bone-metastatic cancers (predominantly prostate and kidney), and to pinpoint key factors responsible for aggressiveness and chemo resistance. Our studies involve mouse models of lipolysis, PDX models, 3D culture techniques and patient samples in combination with pharmacological and genetic manipulation, RNAseq and proteomic technologies. Our ultimate goal is to provide translational insight into advancing current treatment options for bone-metastatic disease.

Kristen Purrington, Ph.D., MPH
Associate Professor
Department of Oncology
Wayne State University
purringk@karmanos.org

My research focuses on the impact of tumor biology and microenvironment on racial disparities in clinical outcomes for African Americans with cancer. Specifically, I have three main areas of active research: (1) Characterizing clinically relevant molecular and immune profiles of breast and colorectal tumors in AAs, (2) understanding heritable susceptibility to breast, prostate, and colorectal cancer among AAs, particularly characterizing variants of uncertain significance in known cancer susceptibility genes, and (3) understanding the role of variability in estrogen receptor protein expression in racial disparities in ER+/HER2- breast cancer survival.
and treatment with endocrine therapy. I am also interested in the combination of genetic factors and traditional epidemiologic risk factors, such as hormonal exposures and mammographic density, and how these may explain risk, biological changes in tumors, and ultimately survival. Much of my research utilizes data and biospecimens from the Detroit Research on Cancer Survivors (ROCS) cohort study, an NCI-funded prospective cohort study of African Americans recently diagnosed with breast, prostate, colorectal, and lung cancers.

Ramandeep Rattan, Ph.D.
Adjunct Faculty
Department of Oncology
Henry Ford Hospital
rrattan1@hfhs.org

Immunometabolism is an emerging field that investigates the interplay between immunological and metabolic processes. Metabolic processes regulate immune cell response in healthy individuals as well as during various disease states, including cancer.

1. Immunosuppressive myeloid cells are a key cause of orchestrating an immunosuppressive microenvironment in ovarian cancer. The ovarian cancer environment induces metabolic reprogramming of myeloid cells and induces a ‘hyper-immunosuppressive’ ability in these cells. Current investigations are focused on (i) identifying the exact metabolic adaptation that facilitates the hyper-active state of immunosuppressive myeloid cells and (ii) targeting of myeloid cell metabolism by use of mitochondrial and glutamine inhibitors.

2. Lifestyle-associated pathologies have an immense effect on the outcome of cancer patients. Unhealthy diets and lifestyles cause chronic metabolic inflammation that can have long-lasting immune reprogramming. We are investigating how the dietary intervention of caloric restriction and time-restricted feeding results in an improved immune response, specifically by regulating intracellular macrophage metabolism and polarization. We are testing various forms of caloric restriction approaches in obesogenic and aging models.

3. The development of chemoresistance is the biggest hurdle in the treatment of recurrent ovarian cancer. We are investigating the metabolic adaptations acquired by chemoresistant cancer cells, focusing on energy metabolism and mitochondrial STAT3.

Sheryl Roberts, Ph.D.
Assistant Professor
Department of Oncology
sherylroberts@wayne.edu

The Roberts Lab is studying how molecular and chemical tools in cancer biology can solve problems for patients with cancer. Our focus is to selectively label malignant growths in animal models of disease to allow for their detection, treatment, and removal. Our goal is to be able to map out and observe imaging/therapeutic response via optoacoustics, which will ultimately benefit patient for early detection of cancer and non-invasive monitoring. My laboratory offers to enhance your skills, performance and it is open to a wide variety of
opportunities to kick-start your career. Our team fosters diversity, equity, and inclusive efforts, and I am committed to individualized mentoring that is design to encourage independence and proactiveness approach.

Lab website: [https://imaginglab.org/](https://imaginglab.org/)

Minhong Shen, Ph.D.
Assistant Professor
Department of Pharmacology and Oncology
Wayne State University
Minhong.shen@wayne.edu

Our lab is interested in understanding the interplay between tumor microenvironment (TME) remodeling and cancer treatment resistance. TME plays pivotal roles in cancer treatment responses. Our long-term research goal is to provide insights into the following two questions: 1) How does TME contribute to cancer treatment resistance; and 2) How could we remodel TME to enhance therapeutic responses. To this end, we are currently focusing on the following three areas: a) Investigate the role of extracellular matrix protein Tinagl1 in TME remodeling and evaluate its therapeutic potential; b) Identify novel candidates that are involved in TME modeling and the consequent treatment responses with high-throughput screening platforms we have established; and c) Develop new TME remodeling strategies to enhance cancer treatment responses by focusing on targeting protein-protein interaction, delivering antisense oligonucleotides, and using Adeno-associated virus mediated CRISPR/Cas9 system.

Nerissa Viola, Ph.D.
Associate Professor
Department of Oncology
Wayne State University
violan@karmanos.org

Research in my group focuses on the development of novel, quantitative imaging agents to interrogate tumor biology. These agents will be examined for their potential to monitor and visualize changes in molecular events during tumorigenesis and after treatment. Our long-term objective is to clinically translate these probes to support precision medicine initiatives. Our work has and continues to lead toward the development of PET radiotracers specific for oncogenic biomarkers and immune signatures (i.e. CD3, IFN-γ, CD8, EGFR, HER2, HER3, tumor acidosis, PSMA, CA19.9).
We are interested in metabolism. We aim to unravel the metabolic basis for complex human diseases such as metabolic syndromes and cancer. Employing advanced research tools ranging from core biochemistry and cell biology assays to genetically engineered animal models, we focus on identifying and characterizing novel and aberrant metabolic activities critical to human pathophysiology. Currently, we are concentrating on the following projects: 1) lipid mobilization and metabolic stress signaling – this DOD-funded project investigates how intracellular lipolysis activates metabolic stress signaling to suppress prostate cancer cell proliferation and tumor aggression. 2) Amino acid metabolism and systemic metabolic homeostasis – this NIH-funded project investigates how hepatic serine and one-carbon metabolism modulate systemic energy production, utilization, and storage in the pathogenesis of metabolic syndromes.

The Wilson Lab studies endometrial cancer, utilizing mouse models to characterize the impact of genetic, epigenetic and metabolic features in the pathogenesis of this disease. Current studies are focused on understanding the role of obesity in promoting endometrial cancer, as well as uncovering molecular mechanisms by which several genetic factors contribute to racial disparities for endometrial cancer. Our goal is to identify novel therapeutic targets such that uterus-sparing treatments can be developed.

Research in my laboratory is to understand how cancer cells develop drug resistance and to develop novel strategies to improve cancer therapies. Our ongoing projects include 1) the regulation of tumor necrosis factor-related apoptosis-inducing ligand (TRAIL) in breast cancer; 2) the role of mitogen-activated protein kinase phosphatase-1 (MKP-1) in platinum resistance; 3) the role of the autophagy pathway in platinum and poly (ADP-ribose) polymerase] (PARP) inhibitor resistance in breast and ovarian cancers; and 4) the regulation of programmed death-ligand 1 (PD-L1) in cancers.
Guojun Wu, Ph.D.
Department of Oncology
Wayne State University
wugu@karmanos.org

The long-term research goals of Wu laboratory at Karmanos Cancer Institute are to decipher the genetic and epigenetic basis for breast cancer initiation, progression, and metastasis and develop a novel therapeutic strategy to target cancer metastasis and overcome chemoresistance. The research projects currently available for Ph.D. or MD students are 1) investigating the functional roles and regulatory mechanisms involved in EMT and metastatic promoting genes; 2) establishing new paradigms for RNA-binding protein in tumor progression and metastasis; 3) screening for small molecules or antibodies that are targeting specific protein or protein-protein interaction responsible for tumorigenesis and metastatic progression; and 4) developing strategies to increase the efficacy of immunotherapy based on unrevealed novel molecular mechanisms in the tumor microenvironment of breast cancer.

Zeng-Quan Yang, Ph.D.
Professor
Department of Oncology
Wayne State University
yangz@karmanos.org

Our research efforts have been focusing on understanding how genetic and epigenetic aberrations cause cancers, and identifying novel therapeutic targets for cancer treatment. In particular, we are focusing on genetic aberrations of histone lysine methyltransferases (KMTs) and demethylases (KDMs) in breast and prostate cancers. Recently my laboratory conducted a metagenomic analysis of KMTs and KDMs in breast cancer and identified associations among recurrent copy number alterations, gene expression, breast cancer subtypes, and clinical outcome. We identified seven KMT/KDMs have the highest frequency of genetic amplification and overexpression in aggressive basal breast cancer. One of them is the amplified in squamous cell carcinoma 1 (GASC1, also known as KDM4C) that was originally identified from the 9p24 amplified region of esophageal cancer cell lines. Our more recent experiments demonstrate that GASC1/KDM4C induces transformed phenotypes in vitro, and regulates a subset of genes involved in breast tumorigenesis. We are currently studying the fundamental mechanism of these KMT/KDMs in driving breast and prostate cancer growth, so that they can be developed as potential novel therapeutic targets for cancer treatment. Another project in our laboratory is aimed at studying the role of endoplasmic reticulum factors in malignancy maintenance and therapeutic resistance of breast cancer.
We have a long-term interest in the roles of two post-translational modification enzymes: histone deacetylase 6 (HDAC6) and ubiquitin-specific peptidase 10 (USP10) in non-small cell lung cancer. Specifically, our research focuses on the roles of HDAC6 and USP10 in the DNA damage response, tumor microenvironment, and metabolism. We are currently using cancer cell lines, syngeneic mouse models, xenograft mouse models, and genetically engineered mouse models to study in vitro and in vivo functions of HDAC6 and USP10 in tumorigenesis.
Alexander Gow, Ph.D.
Center for Molecular Medicine and Genetics
Wayne State University
agow@med.wayne.edu

Dr. Gow studies the role of the unfolded protein response in neurodegenerative diseases; molecular characterization and regulation of axoglial junction assembly in CNS myelin; molecular characterization of the claudin family of integral membrane tight junction proteins during development in brain, testis, and inner ear using transgenic and homologous recombination in embryonic stem cells; and learning and memory deficits in neurodegenerative disease.

James Granneman, Ph.D.
Center for Molecular Medicine and Genetics
Wayne State University
jgranne@med.wayne.edu

Dr. Granneman studies adipose tissue cell and molecular biology, therapeutic adipose tissue remodeling, single cell functional genomics, molecular biology and biophysics of lipolysis activation, co-evolution of lipid droplet binding proteins and molecular pharmacology of ABHD5, a novel therapeutic target for metabolic disease and cancer.

Lawrence Grossman, Ph.D.
Center for Molecular Genetics and Genomics
Wayne State University
lgrossman@wayne.edu

Dr. Grossman and his lab work on mitochondrial molecular genetics. Mitochondria are semi-autonomous organelles because they have their own DNA and genetic machinery but must cooperate with the nucleus to function. Mitochondria contain about 1500 proteins, most nucleus encoded, and carry out a number of functions, most centrally to provide most of the energy in the cell. So important is proper energy function in that mitochondria turn out to be responsible for, or to be involved in, a growing array of diseases, including many common late onset diseases such as various peripheral neuropathies, cardiomyopathies, and type II diabetes. They are focusing increasingly on mitochondrial disease mechanisms, including genomic approaches to population disease susceptibilities. They are also interested in the evolutionary emergence of an enlarged neocortex, the most highly oxygen-utilizing tissue.
Maik Hüttemann, Ph.D.
Center for Molecular Medicine and Genetics
Wayne State University
mhuuttema@med.wayne.edu

Our team studies mitochondrial function using genetic and biochemical approaches. We focus on two key components of the mitochondrial oxidative phosphorylation machinery, cytochrome c oxidase (COX) and the small electron carrier cytochrome c (Cytc). COX is the terminal enzyme of the mitochondrial respiratory chain, “burns” the oxygen we breathe to water, and pumps protons across the inner mitochondrial membrane generating the mitochondrial membrane potential, which is utilized by ATP synthase to produce energy in the form of ATP. Cytc has two distinct functions: it delivers electrons to COX, but it also participates in programmed cell death (apoptosis).

The overall goal of our work is to understand the regulation of COX and Cytc in normal and disease conditions. This regulation in turn affects energy production, free radical generation, and apoptosis. Research topics of the Hüttemann group under investigation include 1) cell signaling pathways that act on COX and Cytc, which pathways are often dysregulated in human diseases; 2) lung cancer; 3) neurodegenerative diseases; 4) gene regulation of COX subunit isoforms; and 5) novel strategies to boost mitochondrial function as a future treatment for diseases that manifest themselves in a lack of energy and increased cell death.

Stephen Krawetz, Ph.D.
Center for Molecular Medicine and Genetics
Wayne State University
skrawetz@med.wayne.edu

His group has published over 200 manuscripts detailing the regulation of gene expression by chromatin structure emphasizing human spermatogenesis and its application to personalized medicine. The interaction of genome structure and function continues to be highlighted throughout his research program. Over the last two decades, his group established that the fitness of the paternal contribution reflects the relative diversity of sperm RNAs that continually responds to the environment. They showed that at fertilization the spermatozoon delivers a cadre of unique RNAs to the oocyte. These RNAs may provide an essential component to early paternal genome reprogramming acting as genetic and epigenetic impactors of the fetal onset of adult disease. They provide a personalized timestamp of the physical and reproductive health of Dad, providing the opportunity to develop a personalized blueprint promoting the birth and healthy life of his children.
Li Li, Ph.D.
Center for Molecular Medicine and Genetics
Wayne State University
lili@med.wayne.edu

The research program in Dr. Li Li's laboratory is centered on understanding the molecular mechanisms of smooth muscle dysfunction in the pathogenesis of vascular diseases especially aortic aneurysms and atherosclerosis. The ultimate goal of our research is to discover biomarkers for the diagnosis and treatment of aneurysm dissection and rupture and atherosclerosis progression. We recently generated a new mouse aneurysm rupture model that contains a FBN1 mutation found in Marfan patients. Our current research focuses on the discovery of key signal pathways and biomarkers in regulating transcription regulatory network in response to FBN1 mutation and metabolic stress. We are particularly interested in molecular mechanisms of defective ECM-cytoskeleton interaction in inflammation, oxidative stress and osteochondrogenesis during the pathogenesis of vascular diseases using tissue culture and mouse vascular diseases models.

Francesca Luca, Ph.D.
Center for Molecular Medicine and Genetics
Wayne State University
fluca@wayne.edu

My lab is interested in understanding the genetic and molecular basis of inter-individual and inter-population differences in complex phenotypes. We combine evolutionary and functional genomics approaches to study intermediate phenotypes (e.g.: transcription factor binding, gene expression, protein secretion, etc.) and how they are affected by gene-environment interactions. Our research is funded by the NIH.

Roger Pique-Regi, Ph.D.
Center for Molecular Medicine and Genetics
Wayne State University
rpique@wayne.edu

My research group aims to further our understanding of the human genome by developing computational methods and statistical models that integrate large genomics datasets. We tackle questions such as: Where are the DNA sequences that control gene expression in a given tissue or cell-type? What are the chromatin marks that characterize the state of the cell and are associated with these regulatory sequences? How can genetic variation disrupt the regulatory code and affect the molecular processes leading to a disease condition? Much of our statistical work makes use of computationally intensive approaches that are designed to be effective for extracting subtle signals from large and complex data sets. This requires using or developing new advanced state-of-the-art methods in statistics, computer science and signal processing and applying them to comparative genomics, functional genomics and population genetics. In general, we aim to tackle problems where careful analysis seems likely to yield new biological insights.
Shengyi Iris Sun, Ph.D.
Center for Molecular Medicine and Genetics
Wayne State University
shengyisun@wayne.edu

The Sun laboratory has two major focuses on protein degradation in the endoplasmic reticulum (ER) and inflammation. ER-associated degradation (ERAD) is a fundamental quality-control process that recognizes and targets ER proteins for cytosolic proteasomal degradation, central to human health and diseases. In fact, defects in protein maturation and turnover in the ER underlie the pathogenesis of many human diseases and moreover, many viruses co-opt ERAD apparatus to establish infection and escape immune surveillance. However, little is known about the physiological role of ERAD. Recent studies have established that low-grade inflammation may play a causal role in obesity-associated insulin resistance, type 2 diabetes, and other complications. However, how the inflammation and innate immunity crosstalk with metabolic processes to regulate metabolism remains poorly understood. We are interested in the role of Toll-like receptor signaling pathways in regulating pancreatic β cell proliferation. In the Sun laboratory, we use physiological, immunological, molecular and cellular approaches to dissect the molecular processes of inflammation and ER protein degradation in the context of metabolic diseases, thereby producing exciting new insights into disease pathogenesis and treatment.

Jeffrey Tseng, Ph.D.
Center for Molecular Medicine and Genetics
Wayne State University
ytseng@wayne.edu

In our lab, we study protein structure, function, classification and evolution. We use geometric approaches to address biological and medical issues of protein and RNA structures in Structural Biology and Molecular Evolution. We design parallel algorithms to perform large-scale computations on these 3D structures via Beowulf Linux clusters. Our research focuses on protein structure and function by using protein shape analysis. We are also interested in the areas of surface identification, characterization, and classification, geometric and evolutionary matching techniques, disease-associated non-synonymous single-nucleotide polymorphism (nsSNP), structural variations associated with gene duplication and Knot theory applied in RNA structures. Novel areas of research include the use of high-throughput computing technology for generating millions of surface patches to study protein-protein interactions and geometric modeling for drug discovery.
Research in this laboratory is focused on cellular stress responses originated from the endoplasmic reticulum (ER) and/or mitochondria that modulate inflammation and metabolism that are associated with metabolic disease, autoimmune disease, and cancer. The ER is an organelle that is primarily recognized as a compartment for protein folding and assembly, a pool of intracellular calcium, and a site for lipid and sterol biosynthesis. Physiological states that increase protein-folding demand, or stimuli that disrupt protein folding reactions, create an imbalance between the protein-folding load and capacity of the ER. This can cause accumulation of unfolded or misfolded proteins in the ER lumen—a condition referred to “ER stress”. To ensure the fidelity of protein folding and to handle the accumulation of unfolded or misfolded proteins, the ER has evolved a group of signal transduction pathways collectively termed “Unfolded Protein Response (UPR)” to alter transcriptional and translational programs. The UPR is a critical regulator in the initiation and progression of a variety of human diseases. Research projects in the Zhang laboratory include: 1) regulation of hepatic energy metabolism by ER stress-inducible transcriptional activators; 2) roles and mechanisms for the UPR transducer IRE1α in rheumatoid arthritis and lupus; 3) airborne particulate matter (PM2.5)-induced cellular stress responses and their effects on non-alcoholic steatohepatitis (NASH) and type-2 diabetes; and 4) roles of ER lipid-raft proteins and UPR transducers in breast cancer malignancy maintenance and therapy resistance.

The major accomplishments by the Zhang lab include: 1) revealing the liver-enriched stress sensor CREBH and its functions and molecular mechanisms as a key transcriptional regulator of energy metabolism under the circadian clock or metabolic stress; 2) defining the pathophysiological roles and mechanisms for the primary ER stress sensor IRE1α in B cell development, hepatic lipid metabolism, and macrophage inflammation; 3) uncovering stress mechanisms by which air pollution induces non-alcoholic fatty liver disease (NAFLD), and revealing that the liver is a major target organ of fine airborne particulate matter PM2.5 that is responsible for air pollution-induced NAFLD and Type-2 diabetes in non-obese individuals; 4) identifying the ER lipid-raft protein ERLIN2 as an unconventional oncogenic factor that facilitates luminal breast cancer cell cycle progression, lipid metabolism, and aggressive malignancy.

Dr. Zhang’s research interests are in metabolism in health and disease. Metabolic syndrome is becoming a public health burden. The Ren Zhang lab identified two novel metabolism regulators: 1) lipasin and 2) MNADK (later known as ANGPTL8 and NADK2, respectively), both being encoded by previously uncharacterized genes. The lab studies the functions and mechanisms of ANGPTL8 and NADK2 in mediating lipid and glucose metabolism.
PHARMACOLOGY
Department of Pharmacology Mentors

Andrew Garrett, Ph.D.
Department of Pharmacology
Wayne State University
gm6614@wayne.edu

The Garrett lab is interested in how developing neurons use cell-surface recognition molecules called cell adhesion molecules (CAMs) during neural circuit formation. Our focus is on the process of self-avoidance, which prevents sister neurites from the same cell (“self”) and from cells of the same type (“homotypic”) from becoming entangled with each other. Our model is the mouse retina, where both self- and homotypic-avoidance is important for the orderly organization of the ~100 distinct types of neurons that process visual information.

Ahmed Ibrahim, Ph.D.
Department of Pharmacology
Wayne State University
gt3759@wayne.edu

My research centers on investigating the underlying molecular and cellular mechanisms of Angiogenesis-driven diseases. The focus is primarily on pathological ocular angiogenesis which is the underlying mechanism of a variety of sight-threatening diseases such as Retinopathy of prematurity (ROP), proliferative diabetic retinopathy (PDR), and exudative age-related macular degeneration (wet AMD). Vision loss in these diseases begins in a common biphasic process characterized by initial loss of the preexisting vessel bed followed by hypoxia that triggers a secondary aberrant angiogenesis.

Samson Jamesdaniel, Ph.D.
Department of Pharmacology
Wayne State University
sjamesdaniel@wayne.edu

Redox sensitive molecular mechanisms play an important role in the regulation of cellular damage in acquired hearing loss induced by noise exposure, ototoxic drugs, and aging. Noise is a pervasive environmental stressor, which induces oxidative stress to affect the physiological as well as psychological well-being. My laboratory employs a proteomic approach to elucidate redox sensitive signaling mechanisms/pathways that regulate noise-induced hearing impairment as well as stress response.
Department of Pharmacology Mentors

Christopher Kassotis, Ph.D.
Department of Pharmacology
Wayne State University
christopher.kassotis@wayne.edu

Our lab is focused on identifying and characterizing endocrine disrupting chemicals (EDCs) and mixtures, as well as their potential impacts on human/animal health - with a particular focus on metabolic health (e.g. obesity, etc.). My research uses a mixture of cell culture assays, zebrafish, and sometimes mouse models to identify and characterize molecular mechanisms underlying potential impacts on human and animal health.

Karin List, Ph.D.
Department of Pharmacology
Wayne State University
klist@med.wayne.edu

Proteolysis in the extracellular/pericellular environment is mediated by about 300 different proteases in humans, of which approximately one-third are directly anchored to the plasma membrane. We are particularly interested in a family of cell-surface-anchored proteases: the type II transmembrane serine proteases (TTSPs) and their role in tissue remodeling during epithelial development and carcinogenesis.

Joongkyu Park, Ph.D.
Department of Pharmacology
Wayne State University
joongkyu.park@wayne.edu

The brain stores, retrieves, and erases information every single moment to mediate the organisms’ successful interaction with the environment. It is now widely considered that these functions are mediated by selective strengthening and weakening of synaptic connections (termed ‘synaptic plasticity’). My research goals are to understand those long-lasting synaptic changes at the molecular level. In particular, my main focus is to understand postsynaptic molecular dynamics responsible for memory acquisition, consolidation, and extinction.
The goals of my research are directly relatable to understanding mechanisms that link nutrition and toxicology and may lead to translatable prevention strategies that may limit pollutant-induced metabolic disorders such as diabetes, obesity, and cardiovascular disease especially in at-risk populations. Overall, I aim to investigate biomarkers that link environmental exposures, diet, and metabolic diseases in human populations and to test mechanisms of toxicity using mouse models of cardiometabolic disease.

Our main research objectives are to identify molecular mechanisms underlying the association between bone marrow adiposity and bone-metastatic cancers (predominately prostate and kidney) and to pinpoint key factors responsible for aggressiveness and chemoresistance. Our studies involve mouse models of lipolysis, PDX models, 3D culture techniques and patient samples in combination with pharmacological and genetic manipulation, RNAseq, and proteomic technologies. Our ultimate goal is to provide translational insight into advancing current treatment options for bone-metastatic disease.

The Shen lab is interested in understanding the interplay between tumor microenvironment (TME) remodeling and cancer treatment resistance. Therapeutic resistance that is frequently observed in metastatic cancers is one of the biggest hurdles in cancer treatment. A growing body of evidence suggests that TME plays pivotal roles in cancer treatment responses. Our long-term research goal is to provide insights into the following two questions: 1) How does TME contribute to cancer treatment resistance; and 2) How could we remodel TME to enhance therapeutic responses?
Department of Pharmacology Mentors

Sokol Todi, Ph.D.
Department of Pharmacology
Wayne State University
stodi@med.wayne.edu

Age-related neurodegeneration, including Alzheimer's Disease, Parkinson's Disease and Polyglutamine Diseases (e.g. several Spinocerebellar Ataxias and Huntington's Disease), afflicts millions of people worldwide. General understanding of molecular mechanisms involved in each of these diseases is incomplete, and no cures exist for them. By using a combination of in vitro biochemistry, mammalian cell biology and fruit fly (Drosophila melanogaster) genetics, the Todi laboratory is working to identify and characterize genes important for neuronal homeostasis during normal function and in disease.

Jennell White, Ph.D.
Department of Pharmacology
Wayne State University
jcwhite@med.wayne.edu

Our lab studies the regulation of red cell-endothelial interactions using mass spectrometry and flow-based adhesion assays. We focus on very late antigen-4 (VLA-4), one of the most characterized adhesion receptors in SCD that supports avid interactions between blood cells and vascular cell adhesion molecule-1 (VCAM-1) expressed on the vascular endothelium. VLA-4 can assume multiple activation states that are functionally regulated by signaling pathways to activate adhesion. The overall goal of our work is to understand the regulation of VLA-4 in red cells.
PHYSIOLOGY
Paulo Caceres Puzzella, Ph.D.
Assistant Professor
Department of Physiology
Pcacere1@hfhs.org

Our research aims to identify cell-cell communication mechanisms between epithelial and endothelial cells, and to determine how they contribute to tissue homeostasis and disease. The close relationship between epithelial cells and the underlying microvasculature greatly determines the properties of the tissues and organs where they are located. For this reason, our perspective is that these cell types need to be studied as a functional unit. We believe that bidirectional mechanisms of cell-cell communication between epithelial and endothelial cells maintain tissue homeostasis and contribute to the diversity of epithelia (there are over 150 in the body and possibly an equivalent diversity of microvascular phenotypes). We are interested in identifying novel pathways of cell-cell communication that influence polarized epithelial processes and microvascular angiogenesis, with the ultimate goal of establishing them as contributors to diabetic kidney disease and renal control of blood pressure.

Xuequn Chen, Ph.D.
Associate Professor
Department of Physiology
xchen@med.wayne.edu

Our research focus is to understand, using systems biology approaches, how ER homeostasis is disrupted in diabetes. The ultimate goal is to identify new therapeutic targets to restore ER homeostasis and improve beta cell survival in diabetes. Currently, our lab is working on the following projects with funding support from NIH and other foundations:

- Quantitative proteomics analysis of perturbed ER homeostasis in diabetes
- COPII dependent ER export of proinsulin in beta cells
- Development of quantitative and targeted proteomics technologies

Susmita Chowdhuri, M.D.
Professor
Department of Internal Medicine, Division of Pulmonary Critical Care
schowdh@med.wayne.edu

Dr Chowdhuri is a clinician particularly interested in advancing evidence-based care of sleep-wake disorders for patients in underserved and rural sections of the country. Her research focuses on understanding the pathophysiologic mechanisms of sleep-disordered breathing in high-risk populations, including older adults and prescription opioid users, with a translational goal of developing therapies that stabilize breathing during sleep. She is also interested in clinical outcomes research, including studying the impact of positive airway pressure therapy in patients with COPD-OSA overlap.
Charles Chung, Ph.D.
Associate Professor
Department of Physiology
cc Chung@med.wayne.edu

We are investigating clinically translatable and basic science problems in diastolic function, using integrative muscle physiology methods. Our goals are to 1) determine targets for pharmacologic improvement of cardiac performance and/or 2) determine improved non-invasive measures of cardiac function to assist in care.

Pamela Harding, Ph.D.
Associate Professor
Department of Physiology
Phardin1@hfhs.org

Dr Harding is interested in the role of prostanoids in fibrosis and hypertrophy of the heart. In particular, the role of prostaglandin E2, its receptors and signaling pathways are examined. A cellular and molecular biology approach coupled with whole-animal physiology is used to elucidate the role of these molecules in regulation of the cell cycle, the signaling pathways and downstream transcriptional events that lead to hypertrophy and whether pharmacological inhibition or gene knockout of these pathways reduces the end-organ damage in response to hypertension or myocardial infarction. A number of state-of-the-art techniques are employed in the laboratory including real time RT-PCR, immunohistochemistry and image analysis, flow cytometry, electrophoresis of proteins, DNA and RNA, gene array and cell culture.

Zhengqing Hu, M.D., Ph.D.
Professor
Department of Otolaryngology
Zh1@wayne.edu

More than 1.5 billion people around the world experience some type of hearing disorder, many of which can be attributed to auditory hair cell loss and dysfunction. In a recent paper, Wayne State University School of Medicine researchers Zhengqing Hu, M.D., Ph.D., professor of otolaryngology-head and neck surgery, and Xin Deng, M.D., former research assistant in otolaryngology, investigated the functionality of newly generated hair cells induced by a previously established epigenetic approach in a deafened adult mouse. Results of the study indicated both new hair cell growth and returned hearing functionality of those new hair cells in cochlear sections. This study is significant because it may open avenues to develop novel methods to restore the function of hair cells to treat hearing loss in humans.
Phillip Levy, M.D.
Professor of Emergency Medicine
Edward S. Thomas Endowed Professor of Emergency Medicine
and Associate Vice President for Translational Science, Wayne State University
Director, Mobile Health Unit Program, Wayne Health
plevy@med.wayne.edu

Dr. Levy is a leading cardiovascular disease researcher who has overseen more than 110 funded studies from various entities since his arrival at Wayne State in 2002 including the CDC, AHA and NIH. He has an h-index of 41 with more than 250 published manuscripts and textbook chapters, and has been an invited lecturer on cardiovascular disease and other healthcare topics more than 300 times. In addition, he has mentored countless medical students, resident physicians and junior faculty members at Wayne State University and beyond.

Jason Mateika, Ph.D.
Professor and Graduate Officer
Department of Physiology
Associate Chair for Research, Department of Internal Medicine
jmateika@med.wayne.edu

Dr. Mateika is a respiratory physiologist who is presently investigating the impact of genetically or spinal cord injury induced reductions in central nervous system serotonin on mechanisms that influence breathing stability and cardiovascular/autonomic function in mice. Dr. Mateika is also exploring if repeated daily exposure to mild intermittent hypoxia enhances the impact of continuous positive airway pressure on co-morbidities linked to sleep apnea in humans with intact or injured spinal cords. Dr. Mateika’s research is presently funded by the National Institutes of Health and Veterans Affairs.

Gil Mor, M.D., Ph.D.
John M. Malone Jr. MD, Endowed Chair and Scientific Director of The C.S. Mott Center for Human Growth and Development, Vice Chair for Research
Department of Obstetrics and Gynecology
gmor@med.wayne.edu

The main objective of our studies is to understand the communication between the maternal and fetal components of pregnancy and how pathogens contribute to the disruption of this crosstalk leading to preterm labor. Specifically, research in my laboratory includes the two following areas: immunology of pregnancy which seeks to understand the communication between the maternal and fetal components of pregnancy and how pathogens contribute to the disruption of this crosstalk leading to preterm labor; and ovarian cancer which seeks understand to the role of ovarian cancer stem cells in the process of tumor formation, recurrence and chemoresistance.
The overall goal of our research program is to understand the molecular and cellular mechanisms that regulate metabolic homeostasis, with the long-term goal of translating basic research findings into therapies for human health. Using novel methods to image fatty acid metabolism in real-time and proteomic and lipidomic techniques, we plan to investigate the mechanisms by which cells sense lipids and maintain lipid homeostasis. A second major project will expand upon the novel biology of ABHD5 interactions with the PNPLA lipase family to investigate the role in fatty liver disease and diabetes.

Patrick Mueller, Ph.D.
Associate Professor
Department of Physiology
pmueller@med.wayne.edu

The goal of the research in my laboratory is to learn more about how the brain controls the heart and blood vessels and therefore, its role in determining arterial blood pressure and organ blood flow. In particular, I am interested in how the brain adapts its control of the cardiovascular system to various physiological and pathophysiological states. Currently, he is examining how levels of physical activity contribute to alterations in neurohumoral control of circulation. In a recent study published by the Journal of Comparative Neurology and covered by The New York Times, he and his collaborators found that inactivity can change the structure and function of the a brain region involved in blood pressure regulation.

Donal O'Leary, Ph.D.
Professor
Department of Physiology
doleary@med.wayne.edu

Dr. O'Leary's research is focused on understanding the neural and hormonal mechanisms which control arterial blood pressure, heart rate, cardiac output, regional blood flow and autonomic nerve activity at rest during stresses such as dynamic exercise and how these mechanisms are altered in heart failure, hypertension and other diseases. He is particularly interested in the interaction between, and mechanisms of action of major cardiovascular reflexes, which control autonomic output and how these reflexes may be altered in pathophysiological states. He has also explored the role of purinergic mechanisms within the nucleus tractus solitarius in cardio-respiratory homeostasis, regional blood flow and peripheral sympathetic nerve activity.
Dr. Ortiz’s team studies new genes and proteins that cause cardiovascular and kidney disease by affecting salt and water handling by the kidneys in diabetic and non-diabetic conditions. Dr Ortiz has adapted and developed a large range of methods to study renal and vascular physiology, and the biology of Na/K/2Cl cotransport by NKCC2 including: Next-Generation sequencing, genomics, proteomics, bioinformatics, single cell RNA seq, multi-photon and live cell confocal imaging, CRISPR, and viral-mediated gene editing in the kidney. Dr Ortiz’s goal is to generate a detailed molecular understanding of kidney function and disease to speed the development of targeted precision medicine therapies to treat cardiovascular and kidney damage caused by obesity and hypertension.

Dr. Palaniyandi’s has three main areas of research that he focuses on:

**Reactive aldehydes and aldehyde dehydrogenase (ALDH) in diabetes-induced cardiac damage:** In our lab, we are planning to determine whether diabetic complications, in particular cardiac damage by hyperglycemia can be reduced by accelerating the removal of these aldehydes by using a novel ALDH activator.

**Mast cells and PKC isozymes in cardiac remodeling of diabetic cardiomyopathy:** We plan to determine whether modulation of PKC isozymes (in particular, PKC beta II) result in the attenuation of cardiac remodeling of diabetic cardiomyopathy.

The primary direction of my research is studying polycystic kidney diseases marked by progressive cyst formation in the renal tubules that ultimately leads to renal insufficiency, hypertension and end-stage renal disease. Recently we identified that the ion channel pannexin-1 causes pathogenic accumulation of adenosine triphosphate (ATP) in the cysts leading to disease progression. We have developed a new method of patch-clamp and imaging in freshly isolated cystic epithelial monolayers and implemented it to demonstrate impaired epithelial sodium channel (ENaC) activity and reveal abnormal ATP release and purinergic signaling in cyst lining cells.
Richard Pilsner, Ph.D.
Professor and Associate Director - C.S. Mott Center
Department of OB/GYN
rpolsner@wayne.edu

The Pilsner lab addresses the interface of environmental epidemiology, toxicology, and reproductive health with a particular emphasis on epigenetic mechanisms. Specifically, our research provides a paternal perspective by delineating the role of sperm epigenetics as a pathway linking paternal preconception environmental exposures to reproductive and offspring health. Such research is critical to understand the paternal environmental determinants of reproductive health, early-life development and future health of offspring. We also recognize that translational research extends from bench-to-bedside, and our current research portfolio echoes this notion with research spanning traditional silos that include both epidemiologic and rodent research.

Jeff Ram, Ph.D.
Professor
Department of Physiology
jeffram@wayne.edu

Dr. Jeffrey L. Ram has diverse research interests relating to biodiversity, reproductive biology, neuroscience, invasive species, and human and environmental microbiology. He studies diverse aquatic organisms both as model systems for reproductive biology and neurobiology and as a way to understand changes taking place in Great Lakes habitats and ecology. These projects have included analyses not only of environmentally significant bacteria at beaches and microorganisms in the ballast water of ships, but also correlations of human oral and fecal microbiomes with disease and behavior, and most recently the detection and quantitation of COVID-19 virus in wastewater.

Jayanth Ramadoss, Ph.D.
Professor
Departments of Physiology and OB/GYN
hh1065@wayne.edu

Dr. Ramadoss is committed to develop strategic ways to improve the lives of women and their offspring. The overall long term goals are five fold: 1) Mechanism discovery: to investigate the role of gestational vascular environment as it relates to pregnancy-adaptations in the etiology of gestational and early life disorders; 2) Biomarker discovery: to develop state of the art non-invasive biomarkers and signature profiles during gestation both in healthy and pathophysiologic states; 3) Therapeutic discovery: to predict, propose, and develop nutritional and pharmacologic therapeutic strategies that will have a practical clinical potential to prevent and/or ameliorate adverse pregnancy pathologies and developmental adaptations; 4) Fetal programing of adult-onset diseases: to investigate fetal programing of adult-onset disease states 5) Strategic improvement of quality of life: to discover means to enhance the developmental environment that may have enduring and life-long health benefits for the offspring.
Nour-Eddine Rhaleb, Ph.D.
Professor
Department of Physiology
Nrhaleb1@hfhs.org

We are interested in the role of vasoactive hormones in the regulation of blood pressure and cardiovascular remodeling under normal conditions and during hypertension. Recently we have focused on an endogenous peptide known as Ac-seryl-aspartyl-lysyl-proline (Ac-SDKP), a natural inhibitor of cell proliferation that is widely distributed in the body. It is primarily destroyed by angiotensin-converting enzyme (ACE), and thus some of the beneficial effects of ACE inhibitors may be due to Ac-SDKP. We are investigating how this substance reduces cardiac and/or vascular scarring in vivo by studying the pharmacology and biochemistry of this molecule, its precursor (thymosin-?4), and its signal transduction cascades. Identifying, cloning and characterizing Ac-SDKP’s receptor(s) is one of our main aims in this mega project.

Robert Wessells, Ph.D.
Associate Professor
Department of Physiology
rwessells@med.wayne.edu

Exercise is a powerful protective factor against many age-related diseases, including heart disease, cancer, and diabetes. Our research focuses on understanding the molecular genetic mechanisms that underlie the benefits of exercise. Using the Drosophila model system, we seek to understand neuronal factors that control exercise behavior, muscular factors that control adaptation to training, and adipose factors that regulate metabolism in response to training. We use lab-specific techniques to induce exercise and study its effect on endurance, speed, flight, and cardiac performance.

Jinsheng Zhang, Ph.D.
Professor of Otolaryngology; Research Educator track, full time
Associate Chair for Research
Director - Laboratory of Tinnitus and Auditory Neuroscience Research

His research interests encompass neural mechanisms underlying noise- and blast-induced tinnitus, tinnitus-related traumatic brain injury, brain neuromodulation with sound, electrical stimulation, light and pharmacological agents to treat tinnitus and related neurological disorders, multi-structure electrophysiological recordings, cross-modality neural plasticity, and auditory prostheses.
Zhibing Zhang, M.D., Ph.D.
Associate Professor
Departments of Physiology and OB/GYN
zzhang@med.wayne.edu

The major focus of my research is to study molecular mechanism of ciliogenesis/spermatogenesis, transcriptional regulation of ciliary genes, and novel functions of mammalian central apparatus proteins.

Current Projects:
1. Dissection of the structural base of MEIG1/PACRG complex in assembling sperm flagella
2. The role of intraflagellar transport proteins in mammalian spermatogenesis
3. Novel functions of mammalian central apparatus proteins, their roles in microtubule/cytoskeleton system and tumorigenesis
4. Transcriptional and post-transcriptional regulation of genes in ciliogenesis/spermatogenesis
Bruce Berkowitz, Ph.D.
Department of Ophthalmology, Visual and Anatomical Sciences
Wayne State University
baberko@med.wayne.edu

The goal of my research is to manage prodromal neuroprotective treatments in neurodegenerative disease, a major missing link in the clinical care of these patients. There is an urgent need for disease-modifying treatment starting at their very onset for a variety of neurodegenerative diseases, such as Alzheimer’s disease and Parkinson’s disease, as well as those linked with visual declines and blindness, including diabetic retinopathy and retinitis pigmentosa. Many of these diseases begin with dysfunction of mitochondria. Yet, current imaging methods cannot measure in vivo abnormal neuronal mitochondria activity, such as oxidative stress, and its treatment response. We have pioneered new, accessible, and patient-friendly imaging methods to address these important technology gaps.

Susanne Brummelte, PhD
Department of Psychology
Wayne State University
sbrummelte@wayne.edu

Research in the Developmental Psychobiology lab focuses on the effects of early adverse life experience on brain development and the subsequent behavioral and neuroanatomical changes in both males and females. We are particularly interested in the consequences of drug exposure such as antidepressants or opioids during pregnancy or the postpartum period and how this affects the mother-infant dyad and long-term outcome of the offspring using rats as the animal model of choice. The research addresses important questions on how exposure to early adverse conditions such as pharmacological treatments can influence maternal care, the maternal brain, and the maturation of the nervous system of the offspring using a number of behavioral, neuroimaging and immunohistochemical techniques.

Alana Conti, PhD
Department of Psychiatry and Behavioral Neurosciences
Wayne State University
aconti@med.wayne.edu

The primary objective of my research program is to investigate the cellular effects of traumatic stress and traumatic brain injury (TBI) on drug action, as it pertains to the neuroplastic response underlying the development of pain and addiction behaviors. Specifically, my lab aims to measure neuroplastic changes structurally, as in dendritic spine formation, dendritic morphology, branching and spine density; behaviorally, as in alterations in pain symptoms, and drug intake/preference; and functionally, as in the activation of molecular pathways that regulate neuronal signaling, which we are doing using rodent models of posttraumatic stress disorder and TBI.
Mark Greenwald, Ph.D.
Department of Psychiatry and Behavioral Neurosciences
Wayne State University
mgreen@med.wayne.edu

Dr. Greenwald is a clinical neuropsychopharmacologist who investigates (1) pharmacological, environmental, and individual difference mechanisms of drug addiction (persistent drug use despite adverse consequences) especially opioids; (2) pain conditions especially chemotherapy-induced peripheral neuropathy; and (3) novel medication and neuromodulation treatments for the above conditions. He serves as faculty advisor to the WSU medical student organization Detroit vs. Addiction, and has mentored many PhD, MD, and MD/PhD students on research projects in his lab.

Arash Javanbakht, M.D.
Department of Psychiatry and Behavioral Neurosciences
Wayne State University
ajavanba@med.wayne.edu

Dr. Javanbakht is a psychiatrist and serves as the director of the Stress, Trauma, and Anxiety Research Clinic (STARC). He is heavily involved in treatment of civilians, refugees, and first responders with PTSD. Dr Javanbakht’s research examines the impact of exposure to war trauma in refugee families, and biological (epigenetic, inflammatory, autonomic) and psychological factors of risk and resilience. STARC also uses art, dance and movement, and yoga and mindfulness in helping refugee families overcome stress. Dr Javanbakht also studies trauma and genetic aging among first responders and impact of exercise on mental and genetic health. STARC is also a national leader in utilization of augmented reality and telemedicine technologies for treatment for anxiety disorders and PTSD. Dr Javanbakht is also a public scholar, and his work has been featured on the CNN, National Geographic, Aljazeera, NPR, Scientific American, Washington Post, Smithsonian, PBS, Science, Lancet, American Psychiatric Association, Anxiety and Depression Association of America, American Academy of Child and Adolescent Psychiatry, Society of Biological Psychiatry, and tens of other media outlets.

Justin Kenney, PhD
Department of Biological Sciences
Wayne State University
jkenney9@wayne.edu

We study the neural and molecular basis for individual differences in behavior. Using adult zebrafish as an animal model, we combine careful behavioral analysis with machine learning, molecular manipulations, pharmacology, and whole-brain activity mapping. We study behaviors like exploration of a novel environment, associative fear learning, and aggression. Our work has important implications for the development of personalized approaches to treating psychiatric and other disorders associated with behavioral perturbances.
Donald M. Kuhn, PhD
Department of Psychiatry and Behavioral Neurosciences
Wayne State University School of Medicine
donald.kuhn@wayne.edu

Our laboratory studies disease-, trauma- and drug-induced damage to the central nervous system and the neuropsychiatric outcomes of these conditions. We are also studying the role of the gut microbiome in psychiatric (e.g., anxiety, depression, and other CNS disorders (e.g., Parkinson’s disease) using 16S rRNA gene and metagenomic sequencing. Our lab is equipped with high-throughput, automated sequencing instrumentation and robotics for the study of host-microbiome interactions. Our laboratory also has expertise in bioinformatics and analyses of big data of the kind generated in microbiome sequencing. Our laboratories are located in the John D. Dingell VA Medical Center and our VA-based projects involve the study of health conditions that have significant influence on Veteran health to include Gulf War Illness, traumatic brain injury, and substance abuse disorders.

Hilary Marusak, PhD
Department of Psychiatry and Behavioral Neurosciences
Wayne State University
hmarusak@med.wayne.edu

Dr. Hilary Marusak is a tenure-track Assistant Professor in the Dept. of Psychiatry and Behavioral Neurosciences at Wayne State University (WSU) School of Medicine. Dr. Marusak directs the Trauma History Investigation of Neurodevelopment in Kids (THINK) lab at WSU (www.wsuthinklab.com). Her lab incorporates neuroimaging, behavioral, and physiological approaches to understand neurodevelopmental mechanisms leading to anxiety and other fear-based disorders (e.g., PTSD) in children and adolescents. Other research interests include the impact of cannabis and cannabinoids on brain development and mental health, and behavioral (e.g., exercise, meditation) or pharmacological (e.g., cannabidiol, CBD) interventions that target the endocannabinoid system for the prevention and/or treatment of fear-based disorders in youth.
Anna Moszczynska, PhD  
Department of Pharmaceutical Sciences  
Wayne State University  
ei2744@wayne.edu

The research carried out in our laboratory focuses on the role of protein parkin in addiction to methamphetamine, a neurotoxic psychostimulant drug. We investigate whether anti-addictive properties of parkin involve mitochondria, oxidative stress, inflammation, or aberrant ubiquitination of parkin substrates. Our secondary research interest is in attenuation of toxic effects of methamphetamine on the brain that predispose experimental animal and humans to development of Parkinson’s disease. The third research interest in our laboratory is in transposons, pieces of DNA that can “jump” from place to place within a genome. We investigate whether and how transposon LINE-1 mediates methamphetamine neurotoxicity in rat hippocampus. In our research, we employ rats overexpressing or lacking parkin and a variety of behavioral and molecular biology techniques, including drug self-administration, conditioned place preference test, electrophoresis, immunohistochemistry, enzymatic assays, adeno-associated gene transfer, proteomics, and confocal microscopy.

Patrick J. Mueller, PhD  
Department of Physiology  
Wayne State University  
pmueller@med.wayne.edu

Research in our laboratory seeks to understand how structural and functional neuroplasticity alters the brain’s control of the cardiovascular system. Our studies focus on time- and sex-dependent differences in the progression of hypertension during sedentary versus physically active conditions. Currently, we are examining the interactions between brain-derived neurotrophic factor (BDNF) and glutamatergic neurotransmission in driving excessive sympathetic outflow, a key contributor to hypertension and cardiovascular disease.
Dr. Panza’s lab is currently investigating the autonomic control of blood pressure in individuals living with spinal cord injuries (SCI). His lab also investigates the impact of sleep on autonomic function and rehabilitation. Individuals with SCI have two distinct cardiovascular responses called orthostatic hypotension (drops in blood pressure) and autonomic dysreflexia (spikes in blood pressure). Both of these blood pressure responses significantly impact physical function, activities, and restrict participation in society. Currently, the lab is investigating 8 days of mild intermittent hypoxia can improve blood pressure control in individuals with SCI. Additionally, the lab is investigating if the neuroplastic changes in the control of breathing change over time, and if they improve during sleep. Finally, the lab is exploring mitochondrial adaptations following mild intermittent hypoxia, and if it contributes to autonomic dysfunction in this group. Primary measures obtained during our studies include, but are not limited to; heart rate and blood pressure variability, pulmonary function, electrocardiograms, muscle oxygenation, and motor limb function (hand and leg).

Dr. Perrine is an associate professor in the Department of Psychiatry and Behavioral Neurosciences at the Wayne State University School of Medicine. His research interests include preclinical studies on the neurobiology of addiction, neurobiological relationship between traumatic stress and drugs of abuse, and applying advanced imaging techniques to neuroscience. Dr. Perrine actively engages in mentoring graduate and medical students, undergraduates, and postdoctoral fellows, and he values and supports diversity in research. This is evident based on his participation in multiple training-related activities, including sponsoring fellowship (NIH-F30, F31, and F32) and career development (NIH K01) awards, engaging in group programs (for example NIH-T34 MARC at Wayne State University and NIH-UL1 ReBUILDetroit), and serving on trainee travel award selection committees (at WSU and for the Society for Neuroscience and College on Problems of Drug Dependence).
The research in Sadagurski lab focuses on the hypothalamic regulation of metabolism in states of environmental stress, obesity, and aging. The hypothalamus integrates information from the liver, muscle, fat, and other organs, and orchestrates whole-body metabolic homeostasis. We study signaling pathways operating in hypothalamic neurons and glia cells (astrocytes and microglia), which are highly relevant to the control of systemic metabolism and the age-associated changes in the glia-neuron interactions. Our lab employs a multi-disciplinary approach to manipulate brain neurocircuits and nutrient-sensing pathways using cutting-edge molecular, genetics, and metabolic assessments in rodents.

Jeffrey A. Stanley, PhD, is a neuroimaging scientist with over 25 years of research experience in furthering the understanding of neural mechanisms underlying brain development and aging as well as different psychiatric disorders including schizophrenia, at risk populations for schizophrenia, ADHD, and mood disorders. Through the use of different innovative Magnetic Resonance Imaging (MRI) methodologies including Magnetic Resonance Spectroscopy (MRS), functional MRS, quantitative structural MRI and myelin water imaging, Dr. Stanley has made significant contributions from the perspective of brain chemistry, function, myelin microstructure and morphology to the field of Psychiatry. Specialties include:

- Assessing glutamate modulation related to task using functional $^1$H MRS
- Assessing the in vivo myelin microstructure of white matter tracts using myelin water imaging
- Structural MR imaging using T$_1$ and T$_2$ mapping methods
- In vivo $^1$H and $^{31}$P MRS
- Brain development and aging
My laboratory uses multi-modal in vivo neuroimaging techniques in combination with human laboratory models to investigate neurobiological mechanisms underlying psychiatric disorders, especially substance use disorders (SUDs). Specifically, we are interested in elucidating neurobiological predictors of drug self-administration with the overarching goal of identifying novel treatment targets. Recent research in my lab has focused on stress and neuroinflammation among cigarette smokers and individuals with opioid use disorder (OUD). Lab website: Drug Lab (druglabdetroit.com)