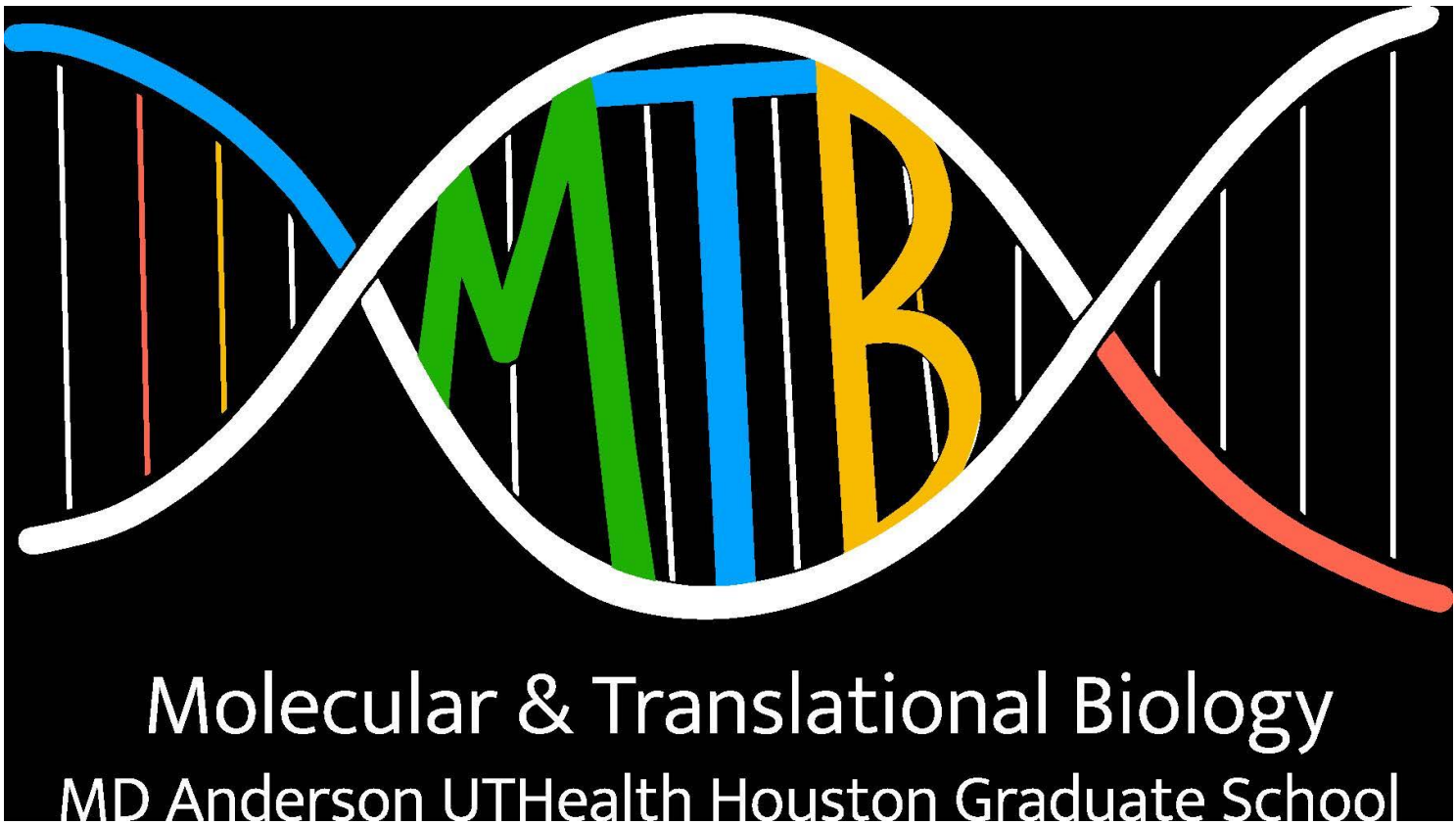
2024-2025

Faculty Seeking Students



2024-2025 MTB Faculty Seeking Students		
Faculty Name	**Research Keywords**	**Seeking Students**
Abe, Jun-ichi	Cardiovascular disease, onco-cardiology, aging	PhD
Akimzhanov, Askar	cell signaling, immune responses, post-translational s-acylation	Both
Carmon, Kendra	antibody-drug conjugates, colorectal cancer, cell signaling	Both
Mikhail Bogdanov,	Folding and Assembly of Membrane Proteins; Lipid and Protein Topogenesis in Bacterial and Cancer Cells	PhD
Chen, Zheng (Jake)	Circadian clocks, Small molecule modulators , Metabolic disease, Aging	Both
Cheng, Xiaodong	intracellular signaling, cAMP signaling, cancer, diabetes	Both
Cordero-Morales, Julio	sensory receptors, ion channels, somatosensation	PhD
Dash, Pramod	memory formation, memory maintenance, hippocampus	PhD
DeBerge, Matthew	Inflammation, cardiovascular disease, neurodegeneration	PhD
Dessauer, Carmen	cyclic AMP signaling, heart disease, chronic pain	PhD
Eckel-Mahan, Kristin	circadian rhythms, metabolism, diabetes	Both
Eltzschig, Holger	Hypoxia, Inflammation, Ischemia-Reperfusion Injury	PhD
Evans, Scott	epithelial biology, antimicrobial molecules, translational therapeutics	Both
Farach-Carson, Mary (Cindy)	tissue engineering, extracellular matrix and cancer metastasis	PhD
Frigo, Daniel	metabolism, prostate cancer, signaling	PhD
Fuentes, Natividad Roberto Jr.	lipid biochemistry, advanced fluorescent microscopy, membrane biophysics and lipidomic techniques	PhD
Gorfe, Alemayehu A.	Molecular Simulation, cell signaling, cancer	PhD

2024-2025 MTB Faculty Seeking Students		
Faculty Name	**Research Keywords**	**Seeking Students**
Gurha, Priyatansh	Epigenetics, Gene Regulation and Heart Failure	Both
Jayaraman, Vasanthi	Structural Biology, Synaptic Signaling, Neuroscience	PhD
Kalocsay, Marian	Proteomics, Receptor Signaling, Immunecheckpoints	Both
Kapoor, Ashish	human genetics and genomics, cardiovascular disease, regulation of gene expression	Both
Karmouty Quintana, Harry	pulmonary diseases, RNA processing, lung explant biobank	MS
Kim, KangHo	Metabolism, Liver disease, Transcription	Both
Klegerman, Melvin	Atherosclerosis, ultrasound, liposomes	Both
Kolonin, Mikhail	metabolism, aging, stem	PhD
Konovalova, Anna	Bacterial Cell Envelope Homeostasis	PhD
Kwartler, Callie	nuclear actin; genetic disease; epigenetics	Both
Lee, Dung-Fang	cancer, genetics, iPSC disease modeling	PhD
Lee, Jayhun	parasite survival, immunity, Schistosomes	PhD
Li, Wenbo	Genomics, epigenomics, bioinformatics, long noncoding and enhancer RNAs	PhD
Liu, Yang	autophagy cargo selection, fasting, cancer	PhD
Liu, Junchen	Lipid cancer mouse	Both
Lo, Hui-Wen	Cancer biology, cancer therapy, cancer biomarkers	Both
Milewicz, Dianna	vascular diseases, genetics, molecular pathogensis	PhD
Miller, Rachel	kidney development, birth defects, Wnt signaling	Both

2024-2025 MTB Faculty Seeking Students		
Faculty Name	**Research Keywords**	**Seeking Students**
Mills, Tingting	idiopathic pulmonary fibrosis (IPF), systemic sclerosis (SSc), and acute respiratory distress syndrome (ARDS)	PhD
Moore, Travis	leukocyte adhesion deficiency	PhD
Morales, Rodrigo	prions; amyloids; Alzheimer's disease	Both
Narkar, Vihang	Nuclear Receptors, Exercise, Myopathies	PhD
Pochynyuk, Oleh	kidney diseases, ion channels, homeostasis	PhD
Pickering, Andrew	Aging, Protein maintenance, Drosophila	Both
Serysheva, Irina	calcium signaling, structural biology, neurodegeneration, cancer	
Song, Min Sup	molecular pathogenesis of cancer, aging, and metabolic disorders	Both
Stavoe, Andrea	Neurons, Aging, Autophagy	PhD
Sun, Kai	Lipid signaling, Cancer research, Obesity and Type-2 Diabetes	Both
Tainer, John	Structural Biology, DNA damage responses, Cancer Biology	Both
Thandapani, Palaniraja	Leukemia, tRNA biogenesis, mRNA translation	Both
Tong, Qingchun	obesity, diabetes, brain regulation	PhD
Vasquez-Robaina, Valeria	mechanosensation, ion channels, behavior	PhD
Venkatachalam, Kartik	neuronal bioenergetics, neurodegenerative diseases, neuronal cell biology	Both
Walters, Edgar T.	spinal cord injury, nociceptor memory functions and mechanisms	PhD
Wang, Jun	craniofacial and cardiac development	Both

2024-2025 MTB Faculty Seeking Students		
Faculty Name	**Research Keywords**	**Seeking Students**
Woodward, Wendy	stem cell biology, radiation biology and molecular biology	Both
Wu, Danielle	tissue engineering, endogenous anti-inflammatory pathways	PhD
Wu, Xiaoqin	molecular mechanisms for regulating intracellular and extracellular vesicle trafficking and cell death in both metabolic homeostasis and disease	PhD
Yan, Jiusheng	structure, function and regulation of mammalian ion channels related to pain	Both
Young, Simon W.M.	implantable biomaterials synthesis, cancer immunotherapy	PhD
Yuan, Xiaoyi	inflammation, immune response, Acute Respiratory Ditress Syndrome (including COVID-19 ARDS)	Both

Join the Cardio-Oncology Revolution with Dr. Jun-ichi Abe!
Are you passionate about making a difference in the world of Cardio-Oncology? Join the lab of **Dr. Jun-ichi Abe**, a pioneer in the field, at MDACC.

What is Cardio-Oncology?
Cardio-oncology is an emerging multidisciplinary field that focuses on the cardiovascular management of patients with cancer. This includes the prevention, diagnosis, and treatment of cardiovascular disease occurring as a side effect of chemotherapy and radiotherapy. Both cancer treatment modalities can cause cardiac dysfunction, a major cause of morbidity and mortality in the oncologic population.

The mission of cardio-oncology is not only to treat heart tumors but also to focus on the cardiovascular effects of cancer treatments. It's about understanding how treatments for cancer can impact the heart and vessels and finding ways to prevent or manage these potential side effects. This is a critical area of research and clinical practice, given the increasing number of cancer survivors and the recognition that cancer therapies can have long-term impacts on cardiovascular health.

Why Join Us?
- **Experience**: Dr. Abe has dedicated 24 years to independent research, with a primary focus on the cardiovascular system's signal transduction mechanisms. His expertise is recognized by the National Institutes of Health (NIH), where he has served as a reviewer for 36 Ad hoc study sections and has twice been appointed as a standing member.
- **Editorial Roles**: Dr. Abe holds prestigious positions on the editorial boards of several esteemed cardiovascular journals. These include the Journal of the American College of Cardiology (JACC), JACC-Cardio-oncology, Circulation Research, Arteriosclerosis, Thrombosis, and Vascular Biology (ATVB), Journal of Molecular and Cellular Cardiology (JMCC), Metabolism, Clinical Science, and the American Journal of Physiology. Furthermore, he is the Section Chief Editor for the Cardio-oncology section at Frontiers of Cardiovascular Medicine.
- **Publications**: Since he arrived at MDA in 2014, He has published 55 original articles in esteemed journals like Circulation, JCI, Metabolism, Redox Biology, and JCI Insight, along with 16 invited reviews, 11 editorials, and a book on Cardio-Oncology.
- **Grants**: He has been awarded 6 NIH RO1 grants as a PI, 5 NIH RO1 grants as a co-Investigator, one NIH R35 grant as a co-Investigator, and one CPRIT grant as a co-PI since 2014.
- **Mentorship**: Dr. Abe has trained and mentored numerous graduate students, postdocs, and Assistant Professors, many of whom have gone on to secure independent faculty positions.

At the Forefront of Cardio-Oncology
Currently, we are at the forefront of research investigating the intricate relationship between telomere shortening, mitochondrial alterations, and their collective role in modulating cellular aging and stemness, particularly as induced by chemoradiation and immune checkpoint inhibitors therapies. This line of investigation is critical for understanding the heightened cardiovascular risks faced by short and long-term cancer survivors.

With a foundation built on more than 30 years of immersive experience in the cardiovascular field, Dr. Abe's expertise has been honed under the duress of oxidative, mechanical, and chemical stressors. This extensive and rich background not only fuels his passion but also fortifies his commitment to advancing our understanding of cardiovascular diseases, especially in cancer survivors.

It is with this profound depth of knowledge and an unyielding drive that Dr. Abe endeavors to contribute significantly to the cardio-oncology landscape, striving to unravel the complexities that underpin cardiovascular diseases in cancer survivors and to pioneer interventions that could transform patient outcomes.

Join Us
If you're ready to contribute to the cardio-oncology landscape and help unravel the complexities that underpin cardiovascular diseases in cancer survivors, join us: Sivareddy Kotla (Associate Professor), Jonghae Lee (Postdoctoral fellow), Venkata S. K. Samanthapudi (Senior Research Assistant), Gilbert F. Mejia (Research Assistant), Oanh Hoang (Research Assistant), Louis Antonio Rivera (MS student), Bhumi M. Patel (GRA), and Edgardo Gabriel Sanchez (GRA).

Together, we can pioneer interventions that could transform patient outcomes!

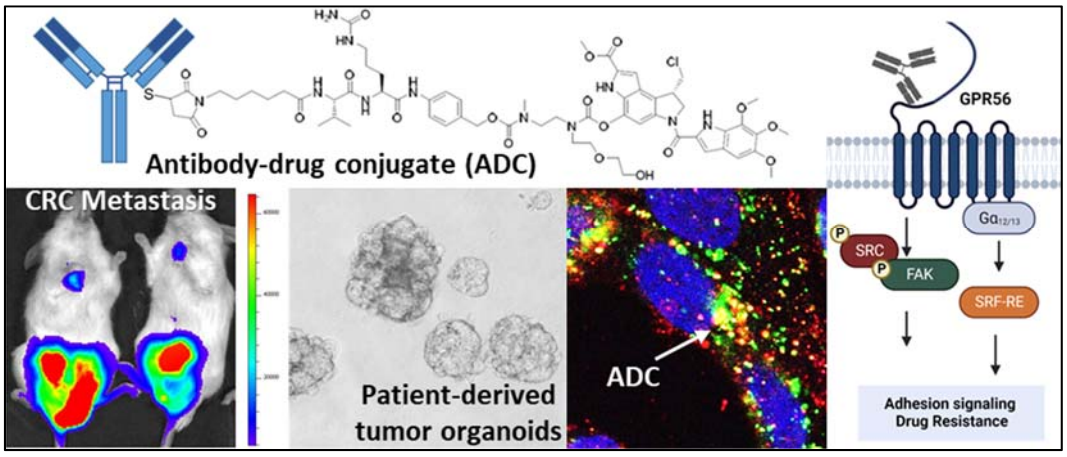
Link to manuscripts
https://www.ncbi.nlm.nih.gov/myncbi/jun-ichi.abe.1/bibliography/public/

Lab website
URL: http://faculty.mdanderson.org/Jun-ichi_Abe/Default.asp

The Carmon Lab

Research Interests:

Despite advances in the treatment of colorectal cancer (CRC), long-term prognosis remains poor for patients with metastatic and recurrent disease, largely due to therapy resistance. Our group is interested in investigating novel proteins and signaling mechanisms underlying therapy resistance, identifying new drug targets, and developing innovative antibody-based treatments to improve and ultimately eradicate CRC.



Projects:

1. Generation of novel antibody-drug conjugates (ADCs) and bispecific antibodies. ADCs are target-guided missiles that link a highly cytotoxic drug with a tumor-antigen targeted monoclonal antibody (mAb) and function to eliminate tumor cells, while minimizing systemic effects. We are generating ADCs against novel targets and evaluating them in patient-derived xenograft models of CRC established in our lab. We are also engineering different formats of bispecific antibodies to simultaneously target two tumor antigens with one therapeutic molecule.

2. Identifying mechanisms underlying cancer cell plasticity and drug resistance in CRC. Cancer stem cells (CSCs) exhibit plasticity or the ability to shift between CSC and non-CSC states to evade therapy. We are identifying novel proteins and pathways involved in plasticity to develop and evaluate improved treatment approaches.

3. Studying the function and signaling mechanism of GPCRs in tumors. Our group is focused on investigating the underlying signaling mechanisms of specific GPCRs upregulated in CRC and other cancers and how they contribute to disease progression. We have developed agonistic mAbs and established collaborations to screen for small molecule agonists/antagonists to examine effects on signaling pathways and function.

Techniques: Cancer cell and 3D tumor organoid culture, antibody engineering, production and drug conjugation, shRNA/CRISPR, cytotoxicity assays, confocal microscopy, immunocytochemistry, cancer cell line and patient-derived xenograft mouse models, non-invasive in vivo imaging, other molecular biology techniques.

Current Lab: PhD students: *Peyton High*, *BS* (MTB Program/TAP; T32/TIPS fellowship, Investing in Student Future's Scholarship), *Cara Guernsey, MS* (MTB program/TAP; Dean's Excellence Scholarship), *Shraddha Subramanian, MS* (Cancer Biology Program/TAP; CRPIT fellowship, Investing in Student Future's Scholarship); Staff: *Zhengdong Liang, MD/PhD,* Senior Research Associate, *Adela Aldana*, Research Associate.

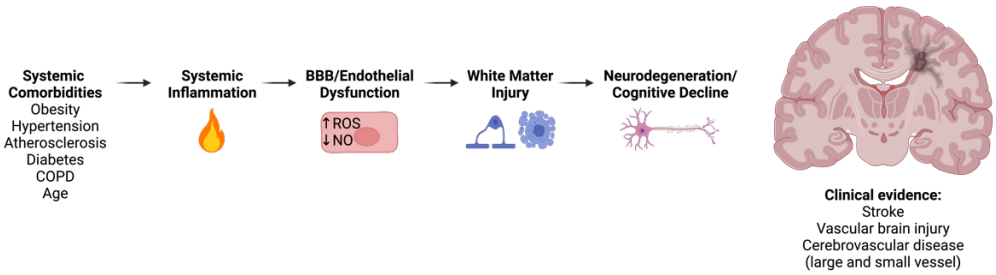
Past Trainees: *Joan Jacob*, *PhD* (former MTB PhD student, now postdoc at Baylor; T32/TIPS fellowship, John J. Kopchick Fellow, Floyd Haar, MD Endowed Memorial Scholarship), *Tressie Posey*, *MS* (former TAP MS student, now Clinical Study Coordinator); *Liezl Francisco, PhD* (former postdoc and CPRIT CTTP fellow, now Regulatory Affairs Specialist, Baylor); *Ashlyn Parkhurst, MD (Resident UTHealth San Antonio); Treena Chatterjee* (Med student, UTHealth); *Carla Godoy, BS* (Med student, Yale).

Contact PI: **Kendra S. Carmon, PhD, Associate Professor and MTB program Co-director,** Kendra.S.Carmon@uth.tmc.edu
MTB Student Contacts: **Peyton High,** Peyton.High@uth.tmc.edu; **Cara Guernsey**, Cara.Guernsey@uth.tmc.edu
-Follow us on X @KendraCarmonLab

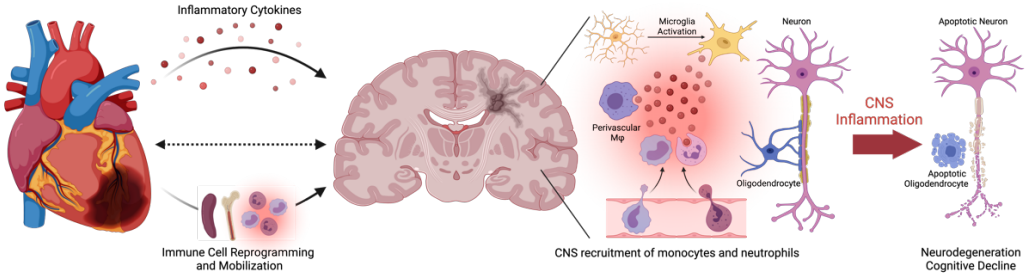
IMPACT Science Center
Lead Investigator: Matt DeBerge, PhD

Research interests:
Inflammation **M**etabolism **P**hysiology for **A**dvanced **C**ellular **T**herapeutics **S**cience **C**enter (IMPACT S.C.) is my experimental team-based approach to science and reflects the scientific focus and mission of the lab in studying the physiology of inflammation, the metabolic signaling that dictates inflammatory activation, and how dysregulation of inflammation leads to disease. The goal is to provide my trainees the mentorship, knowledge base, and resources needed to develop into independent project leaders and achieve their science and life goals.

Projects:
Interfering with microglial neuroinflammation during cardiometabolic disease
The number of individuals living with dementia continues to grow leading to significant morbidity and mortality and increasing the strain on our public health systems. The two most common causes of dementia in the elderly include vascular dementia and Alzheimer's Disease. Vascular dementia lacks effective therapeutics, raising concern that this will only exacerbate the growing epidemic of dementia worldwide. Vascular dementia has stronger associations with cardiometabolic risk factors, including obesity and hypertension. While small-scale prospective studies have found an association between higher levels of systemic inflammation and increased risk for vascular dementia, whether systemic inflammation also reflects neuropathological mechanisms remains unclear. Obesity and hypertension are known to alter myeloid cell abundance and metabolism, the latter a key determinant of myeloid cell reprogramming and function. This raises the possibility that changes in myeloid cells underlies vascular dementia onset and progression.



Inflammatory links between myocardial infarction and vascular dementia
Myocardial infarction is associated with increased risk for vascular dementia, but it is unclear how dementia risk increases after myocardial infarction. In both myocardial infarction and vascular dementia, there is evidence that elevated inflammatory biomarkers are associated with worsened clinical outcomes. Myocardial infarction leads to a systemic inflammatory response, which may contribute to recruitment or activation of myeloid cells, including peripheral monocytes or brain-resident microglia and perivascular macrophages. However, our understanding of the causative roles for these cells linking cardiac injury to the development and progression of dementia is incomplete.

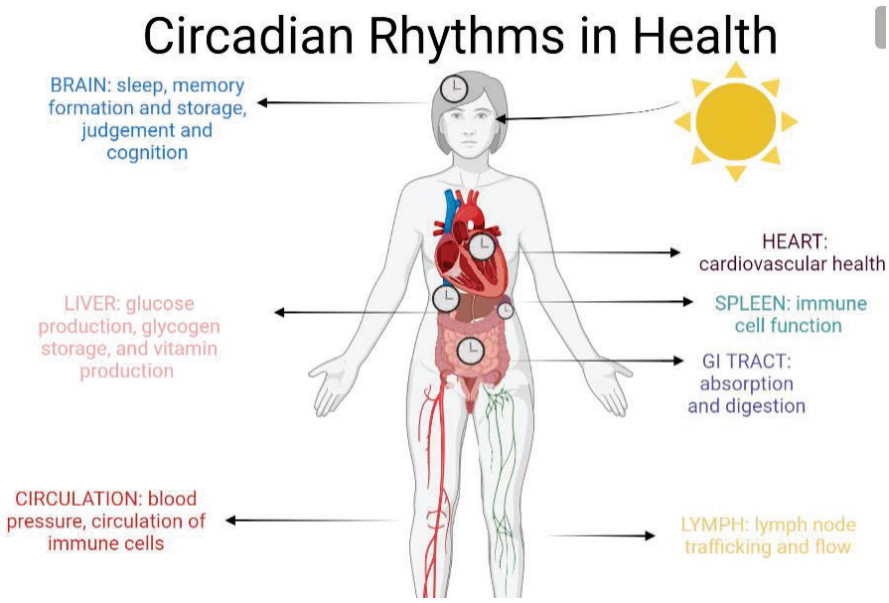


Techniques:
Basic concepts of cardiovascular immunology and neuroimmunology; murine models of heart failure and neurodegeneration; Flow cytometry and cell sorting, ELISA, immunohistochemistry, immunoblot analysis, quantitative RNA analysis.

Email: Matthew.P.DeBerge@uth.tmc.edu
Phone: 713-500-6969
Website: www.impactsc.net

THE ECKEL-MAHAN LABORATORY: CIRCADIAN RHYTHMS IN DISEASE PREVENTION

The goals of my lab center on the role of the circadian clock in health and disease. Circadian rhythms, which are endogenous, self-perpetuating oscillations of 24-hr periodicity, are present in almost all cells of the body. When the circadian clock is disrupted genetically or environmentally, several deleterious outcomes result, including accelerated aging, cancer, and metabolic imbalance. We are trying to understand why circadian disruption produces these effects.



While the central pacemaker of the brain is entrained by light, circadian oscillations in peripheral organs are heavily influenced by other zeitgebers ("time-givers") such as food. When clocks across the body are desynchronized, metabolic disease results. Our current experiments include those designed to reveal which zeitgebers are most important for tissue-specific clock function and the mechanisms underlying their zeitgeber properties. In addition, we are interested in how disrupted peripheral clocks communicate back to the brain and alter neuronal function within the central pacemaker, the suprachiasmatic nucleus, as well as other regions of the CNS.

Current projects in the lab include:

1) the circadian mechanisms by which specific hepatic nuclear receptors prevent liver disease and carcinogenesis

2) mechanisms linking the clock to metabolic function in adipose tissue and adipocyte progenitor cells

3) links between circadian disruption and obesity and insulin resistance

4) mechanisms by which the suprachiasmatic nucleus and hypothalamus orchestrate rhythms in physiology and metabolism

These experiments depend on several mouse models of circadian disruption as well as in vitro approaches.

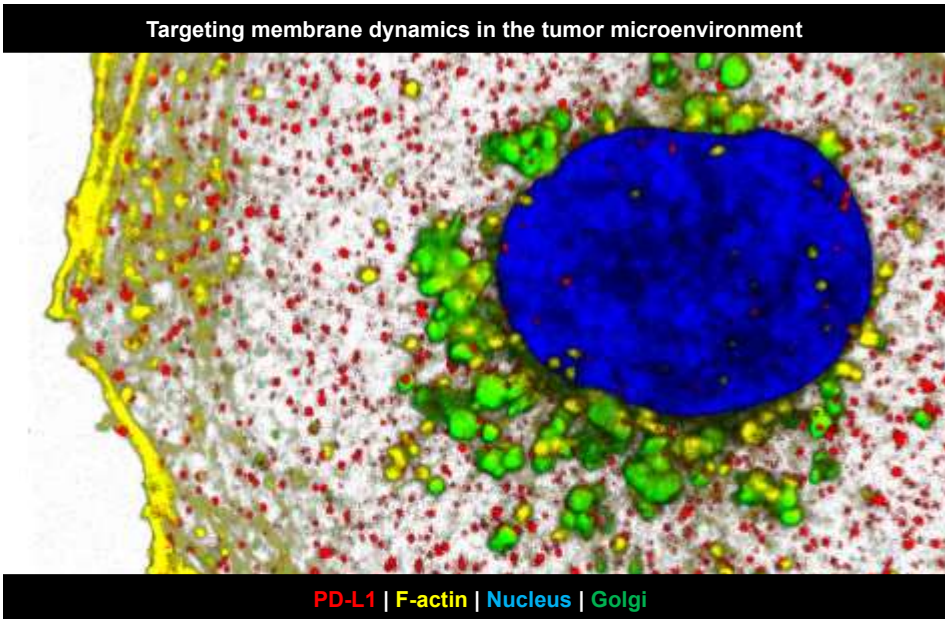
Link to PubMed manuscripts:
https://pubmed.ncbi.nlm.nih.gov/?term=eckel-mahan&sort=date

Come join the great people in my laboratory: Baharan Fekry PhD (Assistant Professor), Rachel Van Drunen (Neurobiology PhD Candidate and Fulbright Scholar), Ruwaida Ahmed (Research Technician), and Sina Noori (Research Technician)

Lab website: https://www.eckel-mahanlab.org/

THE FUENTES LAB: MEMBRANE DYNAMICS IN THE TUMOR MICROENVIRONMENT

Proteins are often credited as the macromolecules responsible for performing critical cellular functions, but lipids have recently garnered more attention as our understanding of their role in cell function and human health becomes more apparent. Lipids serve important functions as signaling molecules and energy substrates and have an often-underappreciated role as membrane structural components. Importantly, membrane spatial dynamics (i.e., biochemical and biophysical characteristics) influence cellular signaling and are altered by disease, such as cancer, or extrinsic factors including diet and environmental exposures. However, the membrane spatial dynamics play in shaping the hypoxic immunosuppressive tumor microenvironment (TME) remains unknown.



Our research program employs dietary intervention strategies, lipid biochemistry, advanced fluorescent microscopy, membrane biophysics and lipidomic techniques to characterize how nanoscale changes to lipids and proteins in the TME influence immunosuppression and other disease processes. Using the TME as a model system we characterize how membrane dynamics act as a non-genetic determinant of cell fate and function. We leverage this information to examine how extrinsic factors such as “healthy” and “bad” diets as well as exposure to environmental chemicals alter membrane structure. Our work will provide greater insight into the mechanism of how membrane structure mediates therapeutic resistance in cancer in order to lay the groundwork for development of novel preventive and/or therapeutic strategies that target membrane spatial dynamics, i.e., membrane therapy. All cells have membranes; therefore, the findings of our research will have a positive impact as the fundamental biological knowledge gained and the development of this membrane therapy approach are applicable to not only cancer but other diseases as well.

Potential Rotation Projects:

1) Identify the molecular mechanism mediating cancer associated fibroblast driven alterations in TME biophysical properties.

2) Determine the functional consequence of altering the biophysical properties of macrophage membranes.

3) Develop a novel membrane targeted therapeutic approach that improves immunotherapy response.

4) Screen for novel intrinsic (genetic) and extrinsic (environmental) modifiers of membrane structure/function.

Lab Website: https://www.mdanderson.org/research/departments-labs-institutes/labs/fuentes-laboratory.html

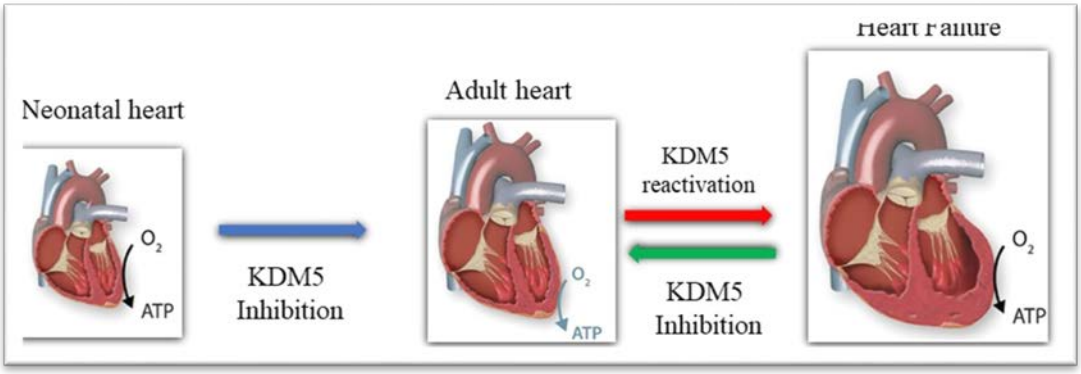
Link to Google Scholar: https://scholar.google.com/citations?user=hZx3S1YAAAAJ&hl=en

Natividad Roberto “Rob” Fuentes Jr., Ph.D., Assistant Professor, Department of Cancer Biology, NRFuentes@MDAnderson.org

Principal Investigator: Priyatansh Gurha, PhD

Research interests: Epigenetic regulation of gene expression in heart

Our goal is to gain a deep understanding of gene regulatory mechanisms to determine their impact on disease, particularly heart failure. In order to accomplish these goals, we employ human tissues, mouse models, and IPSCs Derived Cardiomyocyte models to identify the molecular regulators of gene regulation and their disruption in heart failure. Our most recent research found that KDM5, a member of the histone demethylase family of proteins, is a key factor in cardiac homeostasis. We show that the KDM5 family of proteins affects the maturation and function of cardiac myocytes by controlling cardiac-specific gene programs and mitochondrial OXPHOS genes during development after birth. Furthermore, we demonstrate that, dysregulation of these proteins in adult cardiac tissue leads to heart failure. We are now determining the molecular basis and mechanism of gene regulation for effective targeting of these in the context of heart failure.



We are also interested in identifying and characterizing novel epigenetic modifications, as well as the role of Histone readers and writers in cardiac gene expression. We are using unbiased genomic approaches to determine the role of histone PTM, chromatin regulators, and histone modification enzymes in cardiac function, as well as their impact on aging.

Projects:

- Molecular mechanisms and functions of Lysine demethylase KDM5 in Heart failure.
- Role of histone modifications and epigenetic readers and writers in cardiac function and aging

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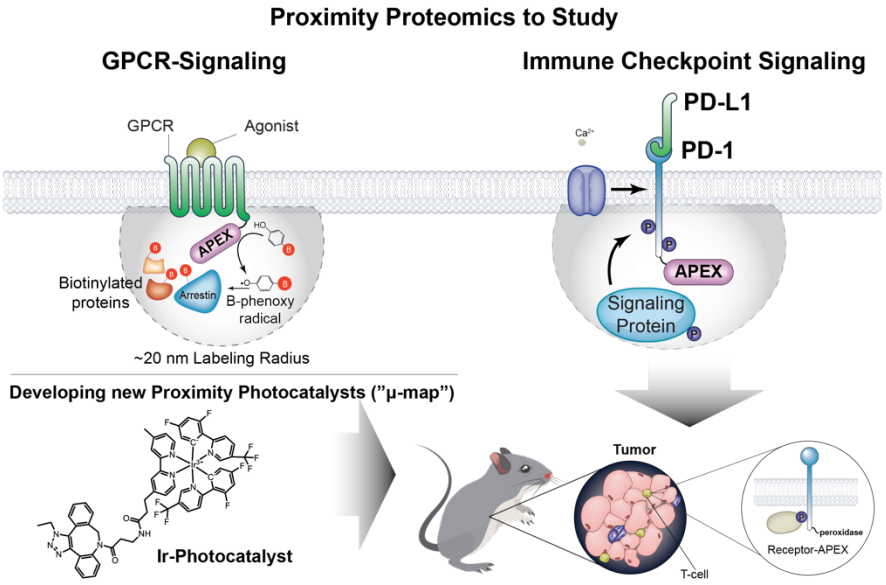
Phone: 713-500-2301

THE KALOCSAY LAB: DECODING IMMUNE RECEPTOR SIGNALING BY PROXIMITY PROTEOMICS

The Kalocsay lab is using **quantitative mass spectrometry** to understand receptor signaling. We have pioneered **proximity sensitive proteomics** to precisely define functional interactions *in situ* at unprecedented temporal and spatial resolution and developed **proximity phospho-proteomics** to precisely follow protein phosphorylation during signaling. We plan to harness these breakthrough technologies to define receptor signaling using experimental models for tumor immune evasion in the future, with the goal of understanding disease mechanisms and developing new therapeutics.

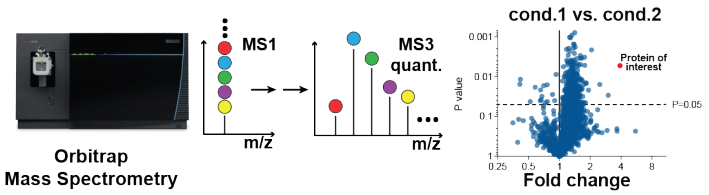
The immune system normally eliminates early malignant cells, thus preventing tumorigenesis. Immune checkpoints prevent overactivation of the immune system, however tumors exploit these checkpoints to evade detection and clearance. Despite enormous clinical importance, we still have an incomplete understanding of which proteins participate in checkpoint signaling, how phosphorylation regulates checkpoint signaling and how uncharacterized (orphan) checkpoint ligands contribute to negative immune regulation.



Our team leverages proximity proteomics and mass spectrometry (MS) to study dynamics and aberrations of signaling processes in the context of immune checkpoints, pushing the envelope of existing MS technology. We hope to elucidate ligand-specific immune signaling and decode phosphorylation in checkpoint signaling with the goal ultimately to better understand how the tumor micro-environment modulates receptor signaling.

Our new lab at MD Anderson is looking for highly motivated master students and Ph.D. students to elucidate cancer-relevant molecular mechanisms in Immune Checkpoint signaling.

We apply top-notch quantitative multiplexed TMT mass spectrometry with newest instruments.



GScholar link: https://bit.ly/3fYk4R4

Lab website:

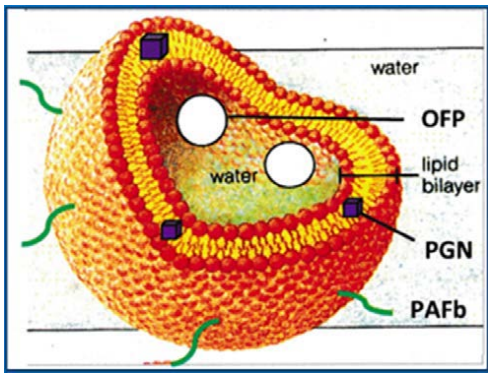
Come join a diverse team and help identify innovative drug targets for cancer therapy.

Klegerman Laboratory

Cardiology Research Laboratory
5110 BBSB
Melvin E. Klegerman, Ph.D.
Professor
(713) 486-2343 **Melvin.E.Klegerman@uth.tmc.edu**

Echogenic Liposome (ELIP) Platform Technology

- Diagnostic/Therapeutic
- Targeted Drug & Gene Delivery
- Ultrasound Controlled Release
- Cardiovascular Applications
- Stem Cell Delivery



Research Opportunities

- Nanomedical strategies for stem cell therapy of cardiovascular diseases
- Ultrasound-based approaches to monitoring and inhibiting atheroprogression
- Biochemical, cellular and immunologic mechanisms of atherogenesis
- Prevention and treatment of lung disorders
- Mechanisms of thrombosis and thrombolysis

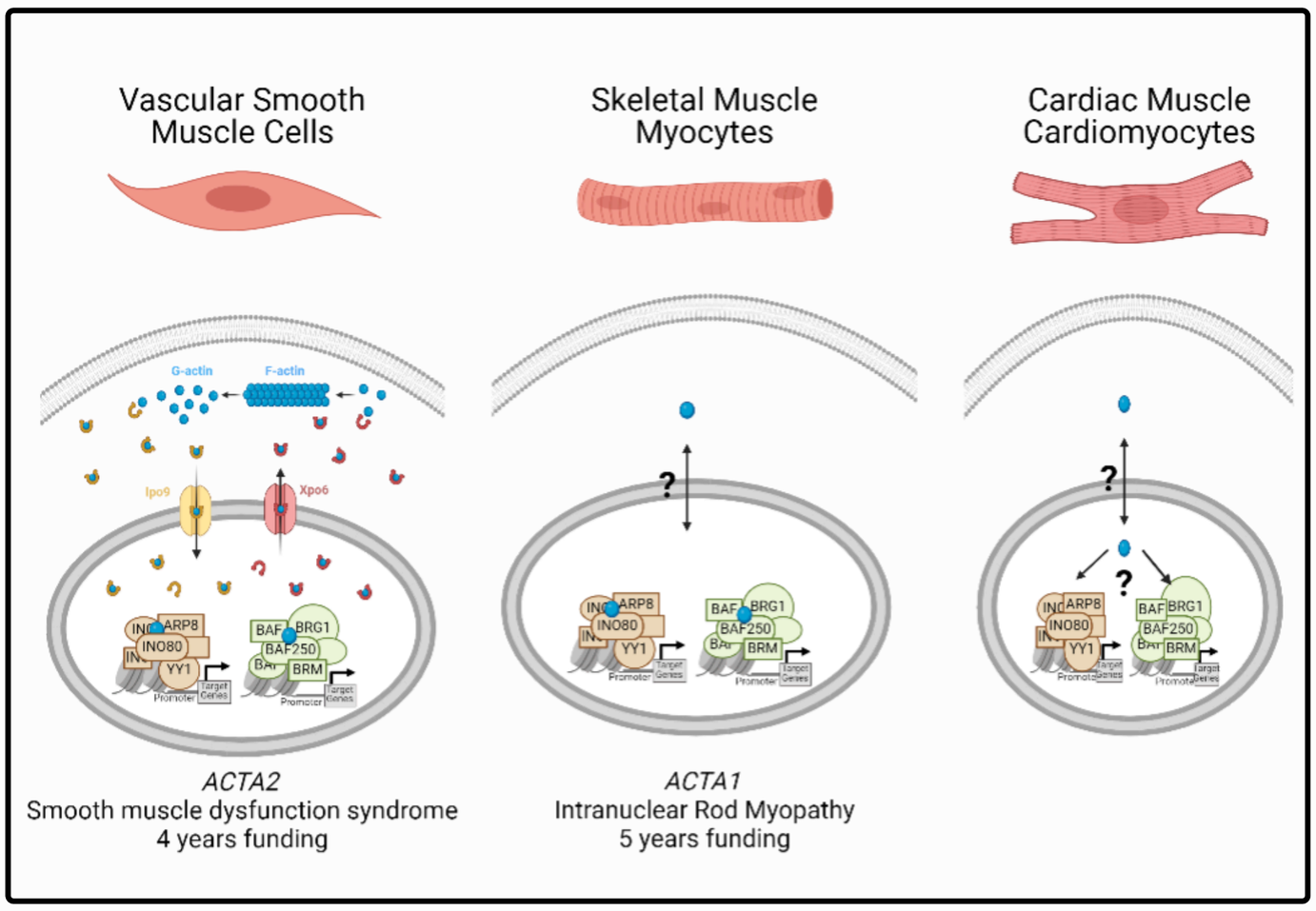
Available Facilities, Equipment and Resources

Wet lab facilities of 5110 BBSB (south campus) and a GLP-compatible production facility on the 6th floor of SCRB 3, including cell culture modules, GC-MS, UV/VIS spectrophotometer, ELISA plate reader, conventional and intravascular ultrasound imaging systems, lyophilizers, liquid chromatography and electrophoresis instrumentation, light microscopes, sonicators, table-top centrifuges, flow cytometer, microplate reader, Beckman Coulter Multisizer 4, Waters HPLC, fluorescent microscopes, Odyssey image analysis system, ultracentrifuge, cath lab facilities for large animal vascular surgeries, ultrasound imaging system for rodent cardiovascular assessments

The Kwartler Lab

Nuclear Functions of Actin

Actin is best known as a cytoskeletal protein that polymerizes and functions in cellular contraction, migration, and other processes. But actin also has nuclear functions: for example, it is a subunit of multiple ATP-dependent chromatin remodeling complexes. Humans (and mice) have six different genes that encode actins, including four that are specific for different types of muscle cells. Our lab previously showed that the vascular smooth muscle-specific actin (ACTA2) is required in the nucleus to promote the complete differentiation of vascular smooth muscle cells. We hypothesize that the other muscle-specific actins are also epigenetic factors that function in the nucleus to promote muscle cell differentiation.



Pathogenic variants in muscle-specific actins cause human disease. We are using these disease- causing variants to better understand the molecular mechanisms of nuclear actins and which nuclear functions are non-redundant between actin isoforms.

We are looking for students to join two projects, each recently funded with multi-year grants. One project looks in depth at how smooth muscle actin is involved in the cell fate specification of vascular smooth muscle cells. We will use single cell transcriptomics during mouse development to assess smooth muscle cell trajectories, epigenomics experiments to determine which alleles are affected by nuclear actin, and test potential epigenetics-based therapies for patients with smooth muscle actin mutations. The second project asks whether skeletal muscle actin affects skeletal myocyte development and whether immature myocytes are the root cause of a particular severe type of skeletal myopathy

For these two funded projects, we are using a variety of model systems including: cultured cell lines, induced pluripotent stem cells (with CRISPR/Cas9 editing), and mouse models.

Mentoring: I am a former GSBS student who has been in your position. As a relatively new PI, I am still actively working at the bench and am very available to my trainees. My aim is to help each trainee grow as a scientist and achieve their own personal career goals. I look for trainees who bring unique perspectives and intrinsic motivation to the lab.

Publications:

[Callie Kwartler's Bibliography]

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Division of Medical Genetics
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713 500 6727 tel I Callie.S.Kwartler@uth.tmc.edu

If you have questions about our science or my mentorship, ask my lab members:

- **Emiliano Esparza Pinelo (Research Assistant)**
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- **Jeison Garcia Serrano (Postdoctoral fellow)**
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- **Kiara Bornes (PhD student)**
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- **Angie Gonzalez (Research Assistant)**
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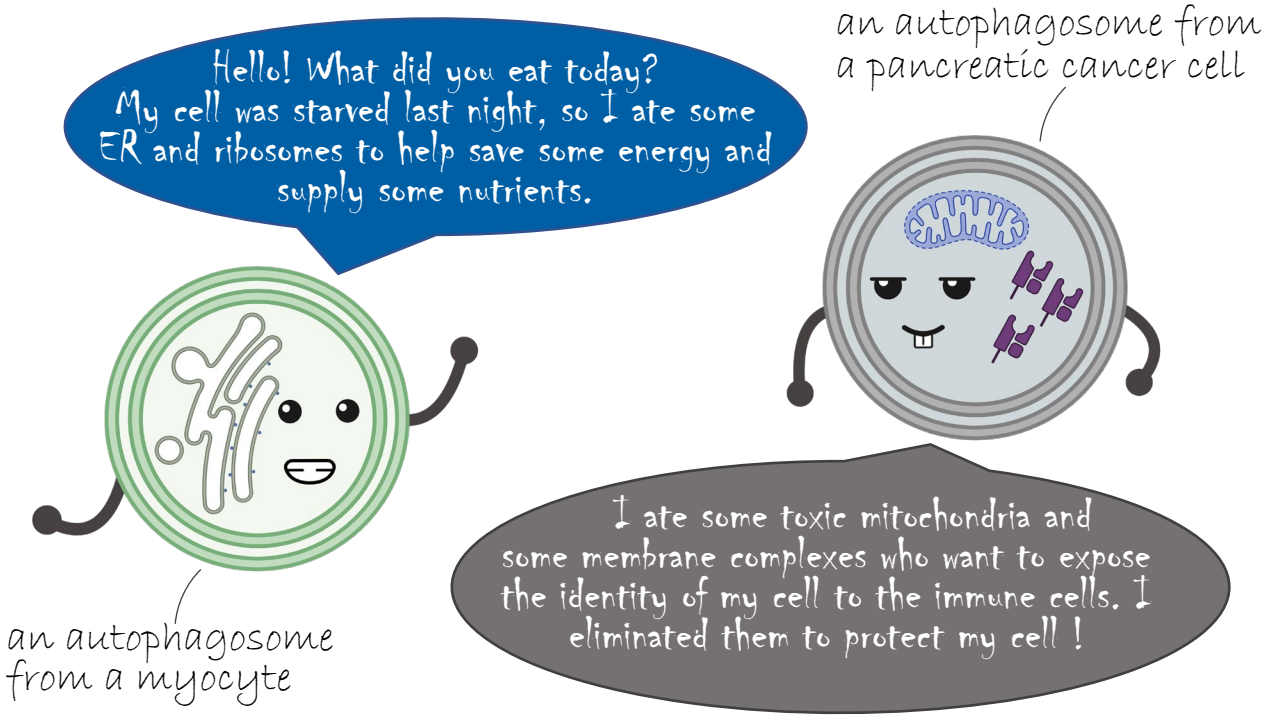
- **Jacob Barham (Undergraduate student)**
jacob.a.barham@uth.tmc.edu

Yang Liu's Lab: autophagy cargo selection in health and disease



Autophagy is a cellular cleaning process to degrade the unwanted cell parts and recycle the nutrients, playing essential roles to maintain cellular homeostasis. During autophagy, the cell parts to be degraded, known as autophagy cargo, are sequestered in autophagosomes before delivery to lysosomes for degradation. In different physiological or pathological conditions, autophagy may play disparate roles through degrading distinct cargo. The focus of our lab is to dissect the molecular mechanisms and (patho)physiological consequences of autophagy cargo selection in physiological/pathological states such as fasting, exercise and cancer development, aiming to better understand the function of autophagy in health and disease.

The current projects in the lab include:

1. To understand the molecular mechanism of autophagy cargo selection during fasting or exercise, and explore how the degradation of the cargo affects skeletal muscle physiology.
2. To understand the autophagy cargo features in cancer cells such as pancreatic ductal adenocarcinoma (PDAC) cells, and explore how the degradation of the cargo impacts tumor cell growth.

The list of publication on PubMed:
https://www.ncbi.nlm.nih.gov/myncbi/1ZimfrtmxdcAu/bibliography/public/

If you are interested, come and talk to us @ MSB4.426 (lab), MSB4.202 (Dr. Liu's office), or email Dr. Liu @ yang.liu.2@uth.tmc.edu

PI: Rachel K. Miller, Ph.D.

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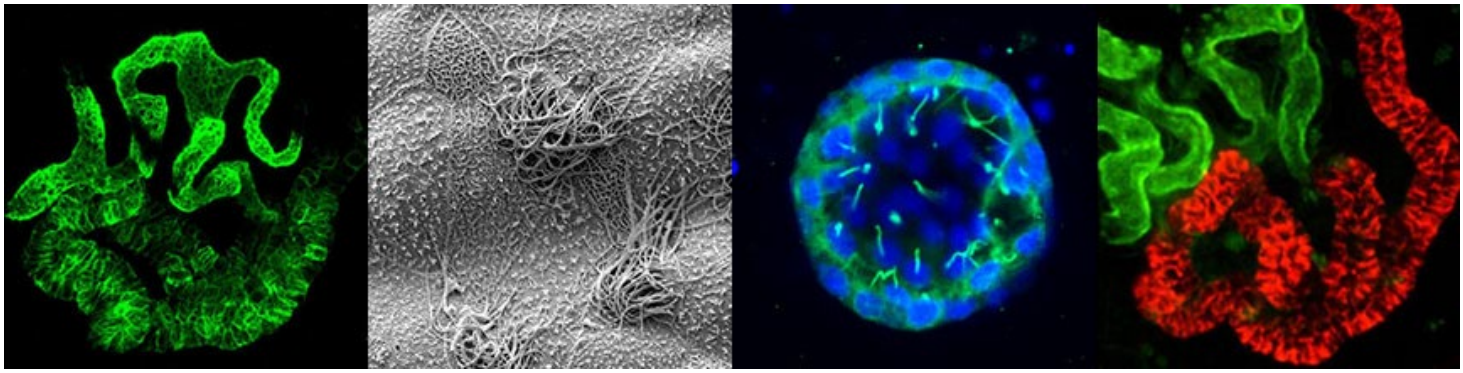
Webpage: https://med.uth.edu/pediatrics/miller-lab/



“Modeling kidney development and disease in frog embryos”

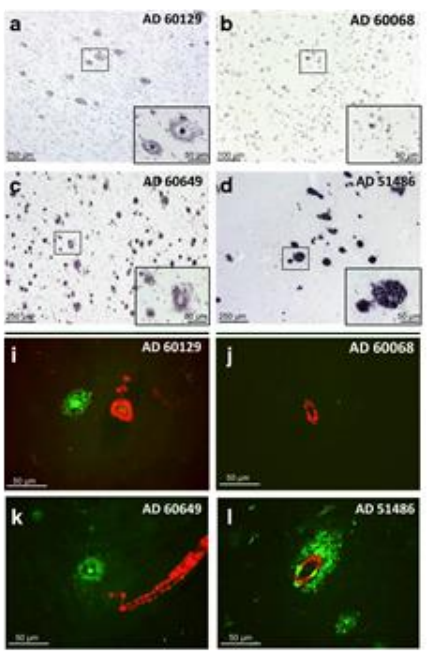
Our overall research goal is to understand the processes that underlie kidney development and how their disruption results in congenital anomalies of the kidney and urinary tract (CAKUT). Malformations of the kidney and urinary tract occur in almost 2% of the world population, representing nearly one-fourth of all birth defects. Because mutations in the genes causing these congenital abnormalities are known in only 14% of cases and often result in the need for transplant, our goal is to understand how these mutated genes disrupt kidney development. Through our use of the frog (*Xenopus*) embryonic kidney, students in our group have made important discoveries related to kidney development (Krneta-Stankic et al. *Cell Rep* 2021) and congenital malformations (Blackburn et al. *Gen Med* 2019). Our trainees have also performed comparative kidney studies using single-cell transcriptomics (Corkins et al. *Kid Int* 2023) and made contributions enabling CRISPR/Cas9 genome editing in the kidney (DeLay et al. *Genetics* 2018). Building on these studies, we aim to understand the cellular processes that drive kidney development.

Environment. We have a highly collaborative laboratory culture, and the valuable contributions of our trainees have been integral to project successes, resulting in a steady record of publication. Collectively, trainees have been awarded a position on an NIH Medical Student training award, a CPRIT undergraduate training award, a Rice Emerging Scholars Howard Hughes award, the Gee Family Legacy Scholarship, the Gigli Family Endowed Scholarship, the Schissler fellowship, the Dean’s Research Award, the GSBS Presidents’ Scholarship, an NIH R01 Supplement, and a Center for Clinical and Translational Sciences TL1 Fellowship. I look forward to advancing the scientific training of students in the future, as I feel it is one of the most rewarding parts of my job.

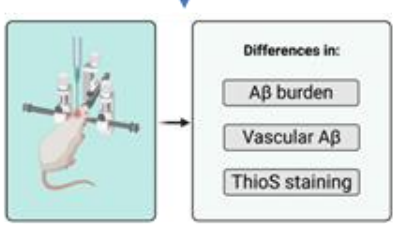
Applying. Our lab is growing! Please get in touch with me if you are interested in working with our group. We wish to recruit one new graduate student.

Rodrigo Morales Laboratory
Department of Neurology
UTHealth

Aβ strains and their role in AD clinical subtypes



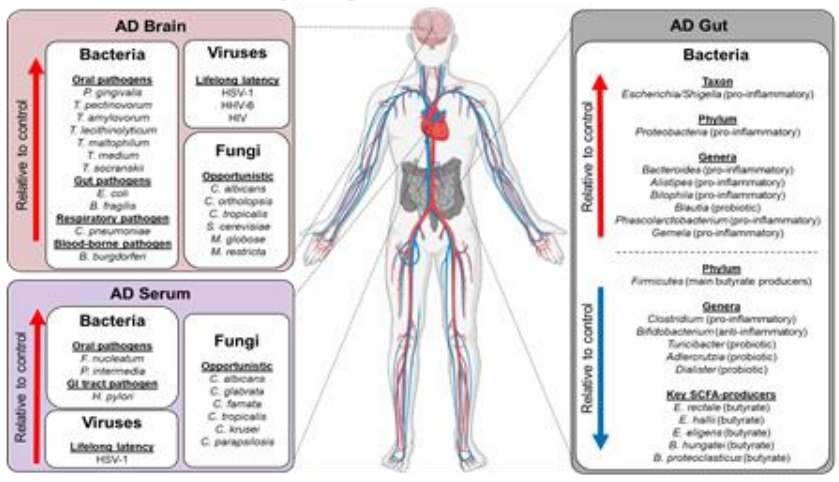
Differential amyloid pathology in patients



Induction of different pathology in mouse models

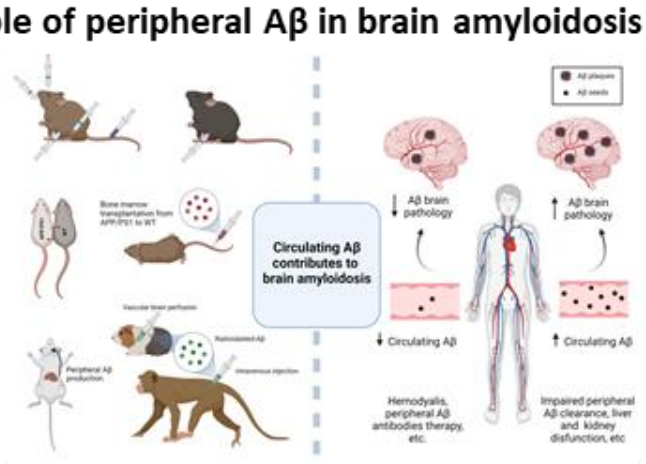
Long-term goal: Aβ strain-specific classification of Alzheimer's disease subtypes. This will lead to personalized diagnosis, prognosis and therapy.

Microbial infections (sepsis, meningitis, COVID-19) as potential risk factors for



Long-term goal: Evaluate the likelihood of bacterial infections to lead to Alzheimer's disease in the long term. This will allow for early interventions to delay or eliminate the chances to get Alzheimer's disease.

Role of peripheral Aβ in brain amyloidosis



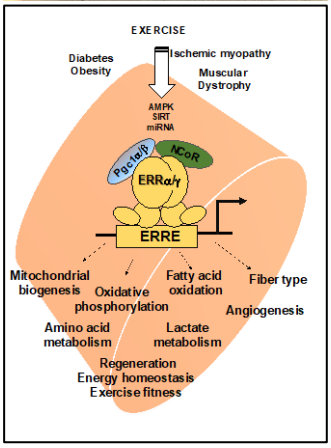
Long-term goal: Understand the contribution of peripheral tissues and blood to Alzheimer's disease. This may open non-invasive avenues for diagnosis and treatment.

Other AD projects at the Morales' Lab

- Role of bacterial amyloids in the progression of Alzheimer's and Parkinson's pathologies.
- Amyloid-contaminated surgical tools and risks for iatrogenic infections.
- Alzheimer's pathology in the eye: mechanistic and diagnostic implications.
- Use of blood from younger individuals as means to decelerate aging: implications for Alzheimer's disease.

Narkar Lab – 'Translational Biology of Exercise Fitness & Muscle Diseases'

Research in our laboratory is focused on nuclear receptor signaling, exercise, muscle fitness and myopathies. We are investigating molecular regulation of (1) muscle function and exercise fitness; (2) muscle mitochondrial metabolism and vascularization; and (3) muscle stem cells and regeneration by nuclear receptors; as well as (4) developing new therapies for diabetes, muscular dystrophy, limb ischemia, and cancer cachexia.



Projects: Following ongoing projects are currently funded by American Heart Association (AHA), Department of Defense (DOD), Hamman Foundation Cardiovascular Research Endowment, and National Institutes of Health (NIH) in our lab, which will potentially lead to treatments for muscle diseases.

Exercise fitness pathways: Exercise is the most effective deterrent of metabolic diseases and myopathies. We are using genetic mouse engineering to identify genes that can increase skeletal muscle fitness, exercise performance and metabolic health. We have generated several transgenic mice, where nuclear receptors such as estrogen-related receptors (ERRs) are selectively activated or deleted in the skeletal muscle. We are measuring the impact of modulating these pathways on exercise fitness and metabolic homeostasis in mice. Further, we are using high-throughput genomic, biochemical, histomorphological, and tensiometric analysis to determine the precise molecular effects of exercise mimetic factors on skeletal muscle architecture and function.

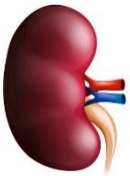
Metabolic diseases and Myopathies: Through our on-going work, ERRs have emerged as transcriptional regulators of muscle mitochondria and vascularization through which they promote fitness and exercise tolerance. In these studies, we are investigating the therapeutic potential of activating ERRs in pre-clinical models of obesity/diabetes, muscular dystrophy, peripheral arterial disease (limb ischemic myopathy), and cancer cachexia. Therapeutic targeting is through small molecules or gene therapy for ERRs developed by our collaborators or in our lab. Ultimate goal of each of these studies is to determine whether mitochondrial and vascular remodeling by ERRs would lead to mitigation of muscle diseases. We are also investigating the role of ERRs in muscle stem, immune cells and FAPS, and relative contribution to muscle regeneration via these cell types in myopathies.

In vitro modeling: Several on-going projects use in vitro muscle systems such as C2C12 cells, muscle stem cells and 3D cultures to explore transcriptional regulation of muscle genome by nuclear receptors, incorporating high-throughput assays such as RNA-seq and ChIP-seq, as well as classical biochemical assays such as kinase signaling, protein-protein interaction and reporter gene assays to identify nuclear receptor transcriptional complexes. These in vitro systems are used in conjunction with our animal models to provide deep insights into how nuclear receptors and their co-regulators orchestrate muscle function.

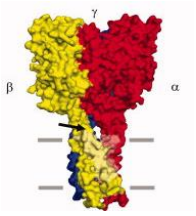
Techniques: Graduate students in my laboratory are trained in a range of techniques including: (1) cellular angiogenesis, myogenesis and respirometry (Seahorse); (2) gene regulation analysis by real time PCR, RNA sequencing and ChIP sequencing; (3) generation and analysis of transgenic mice; (4) skeletal muscle ex vivo tensiometery and histomorphometic analysis; (5) physiological analysis such as treadmill endurance, comprehensive whole-body metabolic analysis via CLAMS, Echo-MRI, and laser Doppler flowmetry; (6) Blood plasma analysis and glucose/insulin tolerance testing in diabetic models; and (7) analysis of disease models of diabetes, muscular dystrophy and diabetes.

Trainees: *GSBS graduate students (current):* (1) Anna DeBruine, B.S. (TAP), (2) Sophia Huang, B.S. (MTB). *Post-doctoral:* (1) Hao Nguyen, Ph.D. (Current); (2) Danesh Sopariwala, Ph.D. (Current); (2) Neah Likhite, Ph.D. (Senior Scientist, Jnana Therapeutics); (3) Pierre Badin, M.D., Ph.D. (Physician Scientist, Montpellier University, France); (4) Vikas Yadav, Ph.D. (Assistant Professor, Jawaharlal Nehru University, India); (5) Antonio Matsakas, Ph.D. (Associate Professor, Reading University, United Kingdom). *Undergraduate trainees:* (1) Manik Kuvalekar, M.S. (Research Associate, Baylor College of Medicine); (2) Patrick Ruggles, B.S. (McGovern Medical School, UTHealth); (3) Annam Sadhana, B.S. (NYU Medical School); (4) Laura Rangel (Universidad El Bosque Medical School, Colombia); (7) Katha Korgaonkar, B.S. (UCSD Medical School); (5) Megha Sheth, B.S. (USC Medical School), (6) Lisa Lin, B.S. (Rice, Pre-Med), (7) Addison Saley, B.S. (Rice, Pre-Med), (8) Eira Mann (UH, Pre-Med).

Contact: **PI:** Vihang Narkar, Ph.D. **Email:** vihang.a.narkar@uth.tmc.edu **Office:** 713-500-3585



Dr. Oleh Pochynyuk's lab



The long-term research in my laboratory focuses on defining physiological roles of different renal ion channels in tubular transport and investigating how their malfunction contributes to pathology at the systemic level. From the pharmacological viewpoint, ion channels are very attractive and promising candidates for drug development. Indeed, pharmacologically-based modulation of activity of ion channels is the backbone of the current treatment of blood-pressure abnormalities, arrhythmias, pain and associated neuropathies, etc. Renal tubule contains numerous channels with poorly defined physiological relevance. Furthermore, some of these channels, such as ENaC, AQP2, ClC-K2/b, TRPV4; exhibit preferential or even exclusive expression in the kidney, thereby minimizing off-target adverse effects when targeted systemically. Thus, through understanding of their roles and unraveling physiologically relevant signaling mechanisms, I expect my research will contribute significantly to the development of innovative pharmacological strategies improving healthcare in the US.

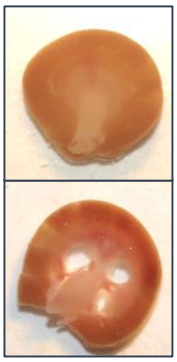
Research interests: Renal (kidney) physiology; ion channels (Na^+, Ca^+, K^+, Cl^-); channelopathies.

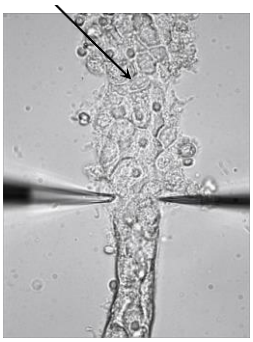
Techniques: patch clamp, $[Ca^{2+}]_i$ and pH_i imaging, confocal immunofluorescent microscopy, metabolic studies.

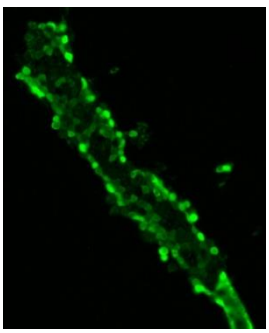
Animal genetic models: global and tissue-specific knockouts; Angiotensin II and salt-sensitive hypertension; polycystic kidney disease, Nephrogenic Diabetes Insipidus.

Why bother joining: 4-5 paper/year; multiple national research awards; opportunities to learn how to write and get fellowships (6 successful career grants for the last 6 years).

Still reading? Contact me: oleh.m.pochynyuk@uth.tmc.edu or simply come: MSB 4.220



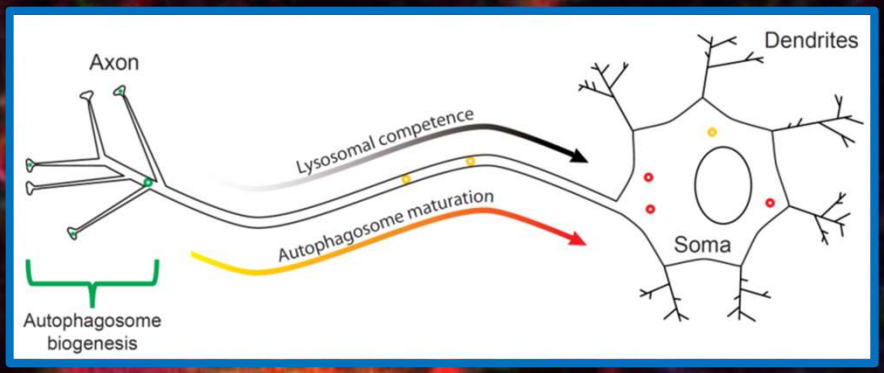


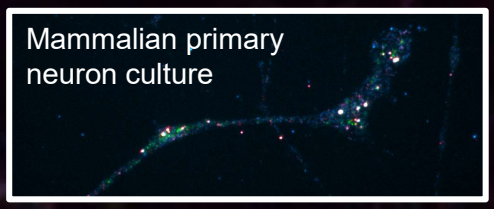


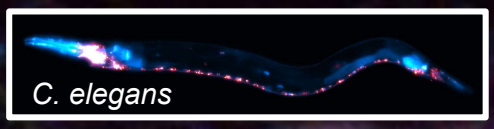
Stavoe Lab

Website: https://sites.google.com/uth.edu/stavoe-lab

How is neuronal autophagy regulated during aging and disease?





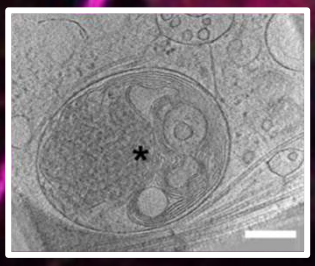


Major Research Questions:

How does autophagy change with age in neurons?

How can we ectopically modulate autophagy in neurons?

Can we extend nervous system healthspan?



Current students:

Heather Tsong, Mya Rodriguez

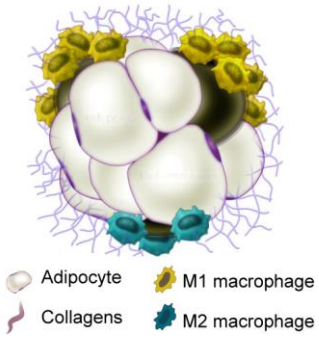
Funding:

afar
american federation for AGING RESEARCH

NIH
National Institutes of Health

SUN LABORATORY

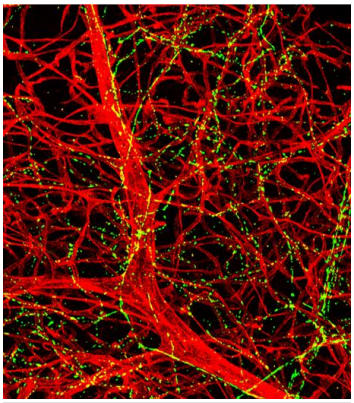
Research Interests: My lab discovers and investigates novel factors that regulate the dynamics of fat tissue remodeling during obesity. The long-term goal of our research is to address the clinical significance of these factors in human obesity, diabetes, cardiovascular diseases and cancer.



Project 1. Hypoxia induced fibrosis and inflammation in fat tissue.

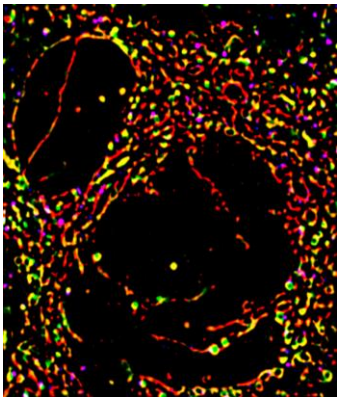
Project 1: ***Hypoxia in obese fat tissue and the metabolic consequences***: In the past years, we have revealed that high fat diet-induced obesity shapes a hypoxic microenvironment that initiates local fibrosis and inflammation in fat tissue. The unhealthy fat tissue further leads to systemic insulin resistance, the hallmark of type 2 diabetes. The related pathological changes are also tightly link to cardiovascular diseases and a certain type of cancer. Current efforts are to identify the downstream signaling pathways that are triggered by hypoxia-induced factor 1 (HIF1) and understand how the events govern the pathological changes, eventually lead to lipid metabolic disorders and insulin resistance.

Project 2: ***Angiogenesis, fibrosis and fat tissue remodeling***: We demonstrated that exercise and cold exposure upregulate pro-angiogenic factor VEGF-A in fat tissue. We further found that VEGF-A-induced angiogenesis ameliorates the pathological changes by suppressing the local hypoxia and stimulating sympathetic innervation in fat tissue. More interestingly, we have revealed that the a proteinase called MT1-MMP facilitates the healthy expansion of adipose tissue in combination with VEGF-A for new blood vessel formation. Moreover, MT1-MMP cleaves collagenous proteins to increase the ECM flexibility in fat tissue. Currently, we apply advanced genetic tools, including doxycycline-inducible fat tissue specific transgenic/knockout models to dissect the mechanisms governing the profound functions of VEGF-A and MT1-MMP in the obese fat tissue.



Project 2. VEGF-A stimulates new blood vessel formation (red) and sympathetic innervation (TH staining, green).

Project 3: ***Dynamics of ER-lipid droplets-mitochondria in fat cells***: Organelle crosstalk is key for metabolic regulations in cellular level. Most recently, we analyzed the dynamics of lipid droplet-associated proteins by Mass Spectrometry. We have successfully identified several novel proteins that translocalize onto lipid droplets and the interface of endoplasmic reticulum (ER)-lipid droplets in response to different cell stimuli. Particularly, we discovered that DRP1 (*left figure, green*) translocates onto ER (*red*) where it promotes the fission of the nascent lipid droplets (*Fig. 3, purple*) from the ER in response to lipid stress. We are now applying state-of-the-art tools and techniques to elucidate the mechanisms governing the functions of the novel factors on the dynamics of lipid droplets and investigating their potential implication in metabolic health.



Project 3. Super-resolution image by n-SIM microscope shows DRP1 (green) targets ER (Red) and releases lipid droplets (purple) in response to lipid stress conditions.

Lab Techniques:

1. Genetic models: Doxycycline inducible, tissue specific transgenic/knockout mouse models. Obese mouse models: *ob/ob* and *db/db* models.

2. Metabolic characterization tools: Euglycemic Clamp; Metabolic cages; Seahorse.

3. Tracking the fine structures and dynamics of organelles: Confocal microscopy, n-SIM super-resolution microscopy; Electron microscopy.

Lab Members: *PI*: Kai Sun, MD, Ph.D. Associate Professor.
Post Docs: Xin Li, Ph.D; Gang Li, Ph.D.
Research Assistant: Shuyue Wang, MS.

Lab Contacts: Dr. Xin Li, Xin.li.1@uth.tmc.edu ; Ext: 713-500-2441 (lab)
Dr. Kai Sun, kai.sun@uth.tmc.edu; Ext: 713-500-3190 (office)

The Tong Laboratory

Neurocircuity for feeding, behavior and metabolism

Major Research Directions

- **To examine key neurons for feeding and metabolism, related to obesity development**;

  Aim to identify neural basis for leptin resistance and come up ways to overcome diet-induced obesity, the cause of the current obesity epidemic

- **To unravel the brain mechanism regulating glucose homeostasis related to diabetes pathogenesis**;

  Aim to identify neural basis for both type 1 and type 2 diabetes: a common neural basis responsible for both diabetes

- **To identify novel neurons and circuits for innate behaviors (aversion, anxiety and aggression) related to psychiatric disorders and mental illness**;

  Aim to elucidate brain mechanisms for co-morbidity between eating disorders and psychiatric disorders

- **To examine the role of glial cells in feeding and metabolic control**

- **Novel creative projects from yourself**

Major techniques

Mouse genetics: Cre-loxP technology to achieve neuron specific manipulations;

Optogenetics and chemogenetics: acute and reversible manipulation of specific groups of brain neuron;

Stereotaxic viral delivery: specific gene expression in highly selected groups of brain neuron;

Fiber photometry and GRIN lens imaging: real time monitoring of neuron activity and neurotransmitter release in behaving animals;

Two photo microscopy: real time neuron activity monitory in behaving animals

Recent Graduates

- **Leandra Mangieri:** PhD 2018, an F31 and UTHealth Best Dissertation awardee; postdoc at UW; now Medical Science Liaison at AbbVie.
- **Ryan Cassidy:** MD/PhD 2019, an F30 and UTHealth Best Dissertation awardee; resident at Vanderbilt, now Assist Professor at Department of Psychiatry and Behavioral Sciences here
- **Jessie Morrill**: PhD 2022, now Assistant Professor at U. Nebraska.
- **Jing Cai**: PhD 2023, Postdoc at NYU.

Contact: Qingchun Tong, PhD Qingchun.tong@uth.tmc.edu