GENETICS & EPIGENETICS PROGRAM

Research Summaries of Faculty Seeking Students

Partial List as of 8/1/2026

See all faculty research profiles on the GSBS website

Swathi Arur, PhD, Genetics, MDA (accepting PhD students)
Richard Behringer, PhD, Genetics, MDA (accepting PhD students)
Andrew Dunbar, MD, Hematopoietic Biology & Malignancy, MDA (accepting PhD students)
George Eisenhoffer, PhD, Genetics, MDA (accepting MS & PhD students)
Yejing Ge, PhD, Cancer Biology, MDA (accepting PhD students)
Jlenia Guarnerio, PhD, Genetics, MDA (accepting PhD students)
Wenbo Li, PhD, Biochemistry & Molecular Biology, UTHealth (accepting PhD students)
Rachel Miller, PhD, Pediatrics, UTHealth (accepting PhD students)
Rodrigo Romero, PhD, Genetics, MDA (accepting MS & PhD students)
Paul Trainor, PhD, Pediatrics, UTHealth (accepting MS & PhD students)
Marianna Trakala, PhD, Genetics, MDA (accepting PhD students)
Aria Vaishnavi, PhD, Cancer Biology, MDA (accepting PhD students)
Peter Van Loo, PhD, Genetics, MDA (accepting PhD students)
Jun Wang, PhD, Pediatrics , UTHealth (accepting PhD students)
Wenyi Wang, PhD, Bioinformatics & Comp Biology (accepting MS & PhD students)
Bingjie Zhang, PhD, Systems Biology, MDA (acecpting PhD students)
Sheng Zhang, PhD, Institute of Molecular Medicine, UTHealth (accepting PhD students)
Xiaotian Zhang, PhD, Biochemistry & Molecular Biology, UTHealth (accepting MS & PhD students)

https://gsbs.uth.edu/genetics-and-epigenetics/index.htm

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Research Categories:

Faculty are listed in one or two categories

Fundamental & Genetic Mechanisms

Swathi Arur, PhD
Richard Behringer, PhD
George Eisenhoffer, PhD
Rodrigo Romero, PhD
Paul Trainor, PhD
Aria Vaishnavi, PhD

Genomic Approaches

Andrew Dunbar, MD
Jlenia Guarnerio, PhD
Rachel Miller, PhD
Peter Van Loo, PhD
Jun Wang, PhD
Wenyi Wang, PhD

Epigenetic Regulation

Yejing Ge, PhD
Wenbo Li, PhD
Bingjie Zhang, PhD
Xiaotian Zhang, PhD

Biology of Human Disease & Cancer

Swathi Arur, PhD
Andrew Dunbar, MD
Yejing Ge, PhD
Jlenia Guarnerio, PhD
Wenbo Li, PhD
Rachel Miller, PhD
Rodrigo Romero, PhD
Paul Trainor, PhD
Marianna Trakala, PhD
Aria Vaishnavi, PhD
Jun Wang, PhD
Wenyi Wang, PhD
Bingjie Zhang, PhD
Sheng Zhang, PhD
Xiaotian Zhang, PhD

https://gsbs.uth.edu/genetics-and-epigenetics/index.htm

Swathi Arur, Ph.D: Professor, Department of Genetics, MD Anderson Cancer Center.
Lab on BSRB 11th Floor.

Students in the Arur Laboratory receive training in multidisciplinary experimental approaches while developing an independent research direction. Our projects span fundamental discovery and disease biology, providing opportunities to work with genetics, quantitative imaging, molecular and biochemical assays, sequencing technologies, mouse models, organoids, and human cancer systems. *The lab currently has one Ph.D student, please contact them for any questions about us!*
https://www.mdanderson.org/research/departments-labs-institutes/labs/arur-laboratory.html

Discover How Signaling Shapes Development, Fertility, and Cancer
How do cells sense their environment and translate that information into decisions about reproduction, development, and disease? Our laboratory combines genetics, cell biology, molecular biology, genomics, imaging, and animal models to uncover how signaling pathways control cell fate and behavior. We work across *C. elegans*, mouse models, organoids, and human cancer cells, allowing students to pursue fundamental biological questions with direct relevance to fertility, birth defects, and cancer metastasis. Graduate students joining the laboratory can contribute to one of three interconnected research areas.

1. How does nutrition influence germ cell development and fertility? Although maternal nutrition has long been associated with offspring health, our laboratory discovered a direct connection between nutrient-responsive signaling and the regulation of female meiosis and oocyte development. We are now investigating how germ cells sense nutritional and somatic signals, how these signals regulate chromosome behavior and oocyte quality, and whether environmental conditions experienced by a mother can influence the reproductive health of her offspring.
Selected trainee publications: Lopez and Chen et al., *Developmental Cell*, 2013; Suen et al., *Nature Structural & Molecular Biology*, 2013; Das et al., *Science Advances*, 2020; Das et al., *PNAS*, 2022; Trimmer et al., *Cell Reports*, 2023; Baek et al., *Cell Reports*, 2025.

Lab day out, 2026 at Meow Wulf.
L-R: Amelia Li, Shin-Yu Chen, Xingxing Yan, Lisa Watson, Ashley Florance, Swathi Arur, Kenny Trimmer, Rares Drula, Han Bit Baek

Talk to them about lab culture.

2. How do signaling pathways control small-RNA production? Our laboratory discovered that environmentally activated signaling pathways directly regulate the small-RNA machinery, including the phosphorylation and activity of Dicer and Drosha. We are determining how signaling changes the localization and molecular functions of these enzymes, why distinct small-RNA populations are generated, and how post-transcriptional regulation controls oocyte development and the oocyte-to-embryo transition.
Selected trainee publications: Minogue et al., *Nature Communications*, 2018; Aryal et al., *PNAS*, 2018; Ortega et al., *Science Advances*, 2024; Newkirk et al., Nature Communications, 2026.

3. How does DICER reprogram cancer cells to promote metastasis? We found that phosphorylation drives DICER into the nucleus and fundamentally changes its biological function. Using genetically engineered mouse models of KRAS- and p53-driven lung cancer, we found that phosphorylated nuclear DICER promotes tumor dissemination and metastatic progression. Rather than primarily regulating microRNAs, nuclear DICER associates with RNA, chromatin, and nuclear protein complexes. These interactions alter chromatin accessibility and the expression of lineage-defining genes. We are now investigating how this signaling-dependent reprogramming creates metastatic potential.
Selected trainee publications: Aryal et al., *Cancer Research*, 2018; Reyes et al., *Science Advances*, 2023.

Richard Behringer, Ph.D.

Professor
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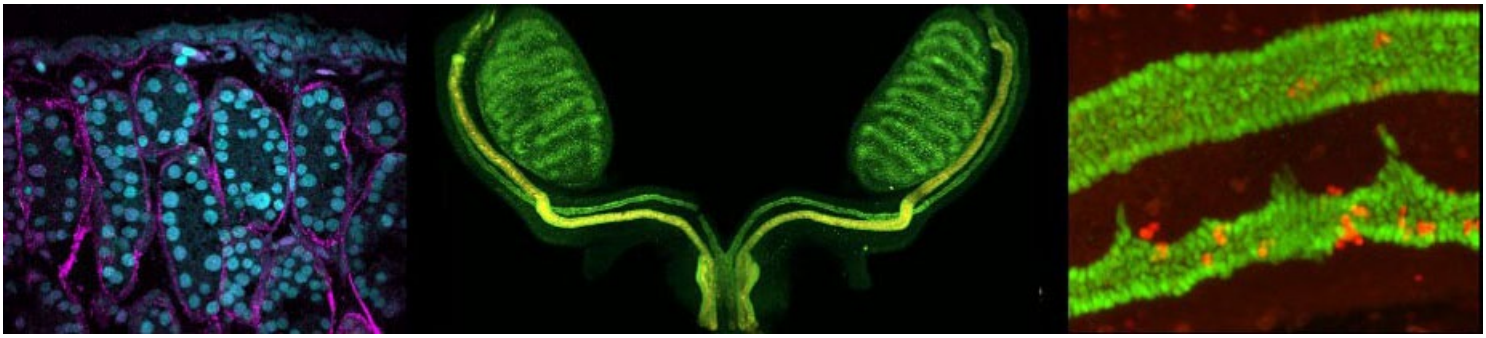
MD Anderson Cancer Center, BSRB S11
rrb@mdanderson.org
https://faculty.mdanderson.org/profiles/richard_behringer.html



Reproductive organ formation and disease

The overall goal of our research is to understand the molecular, cell, and developmental processes that result in the formation of the reproductive organs, the gene regulatory networks that control the differentiation of the male and female phenotypes, and how mutations result in reproductive organ variants. Our primary model system is the mouse but we also have a colony of brown anole lizards to explore these and other processes in reptiles, using CRISPR gene editing. We use cutting-edge microscopy methods, including time-lapse imaging of cell and tissue behaviors and 3D imaging platforms such as microCT, coupled with genomic approaches, including spatial transcriptomics to understand mutant phenotypes. More recently, we are exploring a normal aspect of male differentiation as a novel model for cancer metastasis.

Environment. We have a very welcoming and supportive laboratory culture, appreciating the contributions of each lab member to make innovative discoveries in biomedical research.

Potential rotation projects. Cell scattering during fetal male tissue remodeling. Role of Wilms tumor suppressor gene in male mouse differentiation. Role of p53 tumor suppressor gene in reptiles. Role of anti-Müllerian hormone for oogenesis in frogs.

Apply. Please contact me if you are interested in training in our group.

THE DUNBAR LABORATORY

Understanding how blood cancers evolve — and how the bone marrow environment helps drive that evolution

The Dunbar Laboratory at MD Anderson Cancer Center studies myeloproliferative neoplasms (MPNs), a group of chronic, pro-inflammatory blood cancers that over time, lead to progressive bone marrow scarring (fibrosis) and, in some patients, acute leukemia. Our goal is to understand not only how malignant MPN blood cells evolve and survive with disease progression, but also how they interact with surrounding bone marrow stromal cells, immune cells, blood vessels, and extracellular matrix to reshape the bone marrow niche. Ultimately, we aim to functionally characterize and test identified pathways of interest in order to identify novel drug therapies for these patients.

WHAT WE STUDY:

- **How the marrow "neighborhood" shapes cancer.** We study how malignant blood stem/progenitor cells communicate with bone marrow mesenchymal stromal cells, endothelial cells, and extracellular matrix to promote fibrosis progression and/or transformation to a more aggressive disease, including leukemia.
- **Targetable signaling dependencies.** We investigate how different oncogenic signaling pathways such as JAK/STAT, TNFa/NF-κB, and MAPK cooperate with one another in MPNs, and whether improved targeted inhibition with novel drugs and drug combinations can reverse or prevent disease progression.
- **Genotype, cell state, and treatment response.** We also examine how mutations in epigenetic modifiers such as ASXL1 and EZH2, as well as TP53 cooperate with JAK/STAT oncogenic drivers in MPN to influence cell identity, therapeutic sensitivity, and the path from chronic disease to leukemia.

HOW WE DO IT

- **Spatial biology & single-cell profiling** — high-resolution spatial transcriptomics, single-cell multi-omics, and protein-interaction mapping to reconstruct the architecture of human bone marrow.
- **Mouse genetics & disease models** — reversible and conditional models that let us ask whether a specific oncogenic or microenvironmental signal is truly required for disease maintenance.
- **Primary patient samples & functional assays** — bone marrow specimens, cytokine profiling, cell culture systems, and perturbation experiments to validate mechanisms in human disease.
- **Therapeutic studies** — preclinical testing of emerging agents and combinations, both *in vitro* and i*n vivo*, with close links to clinical MPN research at MD Anderson.

WHAT A GRADUATE STUDENT CAN EXPECT

Projects in the lab are designed to connect a fundamental biological question to clinically meaningful disease. Students can develop expertise across wet-lab and computational approaches, work with both model systems and patient specimens, and collaborate closely with hematologists, pathologists, computational scientists, and technology experts. We particularly value curiosity, intellectual independence, careful experimentation, and a willingness to cross traditional disciplinary boundaries.

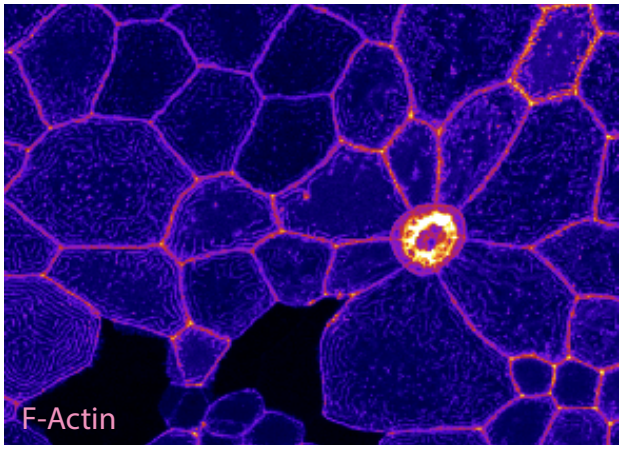
Cancer
UT MD Anderson

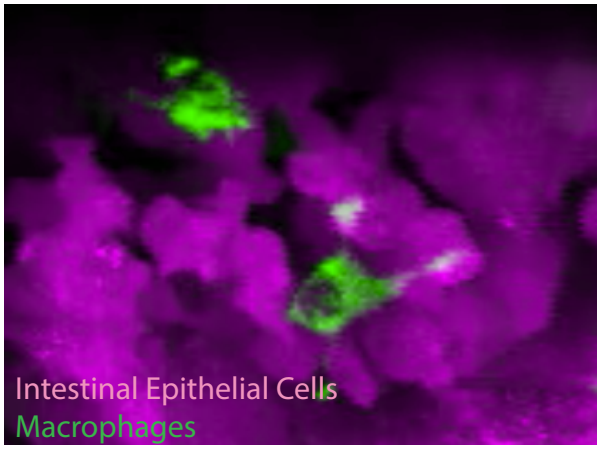
George T. Eisenhoffer, PhD
Associate Professor
Department of Genetics
gteisenhoffer@mdanderson.org

Research Interests
zebrafish development and genetics
epithelial tissue homeostasis
stem cells and regeneration
carcinogenesis and metastasis

Cancer development has long been linked to a mis-regulation of the body's normal homeostatic processes and regenerative responses during wound healing after injury. My laboratory studies the cellular and molecular mechanisms linking the birth and death of cells in living epithelial tissues to better understand how specific genetic changes drive an increase in cell numbers and lead to carcinogenesis. To study cell turnover in a living epithelial tissue, we use the developing zebrafish to rapidly elucidate mechanisms that regulate epithelial cell function under physiological conditions, after tissue damage, and after genetic perturbation. We monitor population dynamics and individual cell behaviors under normal and experimental conditions using high-resolution time-lapse microscopy and integrate these data with single-cell RNA sequencing to gain a clearer picture of how epithelia maintain overall numbers while sustaining a functional barrier.

Our studies have provided mechanistic insight into how localized changes in physical forces are coordinated to remove defective cells from living epithelial tissues (Atieh et al., 2021 Current Biology; Franco et al., 2019 MBoC; Anjum et al., 2024 iScience). We have also interrogated the cell loss-induced signaling events and cellular responses, including inflammatory cell recruitment and epidermal cell proliferation, that drive turnover (Brock et. al., 2019 Nat. Comm; Wurster et al., 2021 Cell Reports; Essien et al., 2024 PLoS Biology). Together, our studies provide an *in vivo* characterization of epithelial cell turnover and create a system to identify new mechanisms controlling tissue regeneration and the changes that lead to cancer formation and progression.



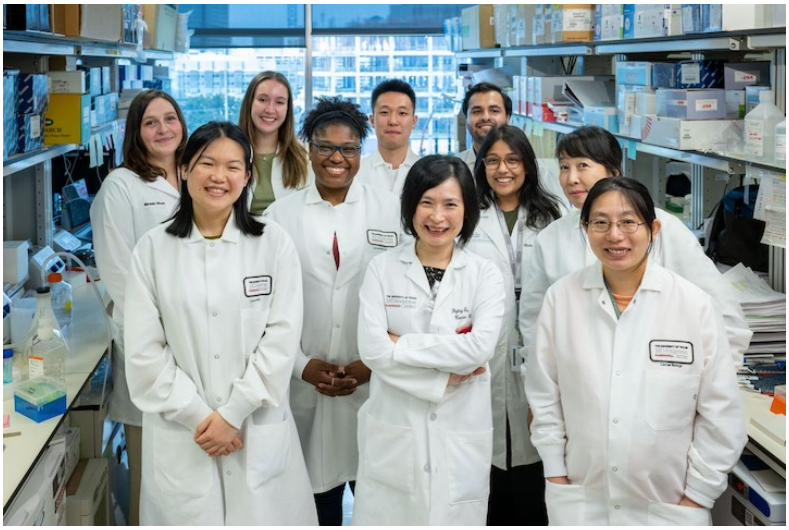


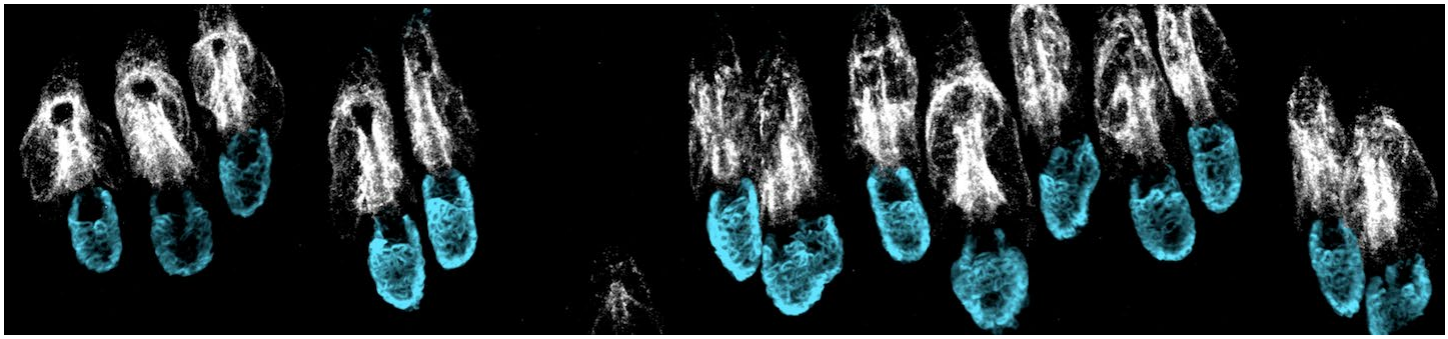
Seeking PhD Student

PI: Yejing Ge, Ph.D.

Associate Professor
Department of Cancer Biology
UT MD Anderson Cancer Center,
Houston TX
Contact: YGe1@mdanderson.org

Ge Lab
Skin Functional Genomics





Defined by golden standards of long-term self-renewal and multi-lineage differentiation, stem cells (SCs) come in different flavors. In mammals, adult SCs are essential units to orchestrate postnatal remodeling and repair damage. Upon stress, SCs often expand their fates and embark on behaviors distinct from their homeostatic patterns, known as plasticity. While plasticity is essential for organismal survival, its derailed regulation poses disease vulnerability to individuals, where SCs are subjected to functional exhaustion frequently observed in aging, or malignant transformation that occurs in cancer (Ge et al, **Nat Cell Biol**, 2016; Ge et al, **Cell**, 2017; Ge et al, **Nat Rev Genetics**, 2018; Ge et al, **PNAS**, 2020; Lyu, Guan, Humphrey et al, 2022 **Genes & Dev**). Research in the Ge lab uses skin as a model, and applies mouse genetics, functional genomics and development biology approaches to dissect molecular mechanisms underlying SC plasticity, and how its deregulation leads to human diseases, including wound repair, cancer, and aging. We recently reported the role of transposons in skin regeneration (Lyu, Kim, Humphrey, Nayak et al, **Cell**, 2024) with strong implications in cancer malignancy.

Come check us out at our website yejinggelab.com We are excited to have passionate individuals join our team!

Our Core Values

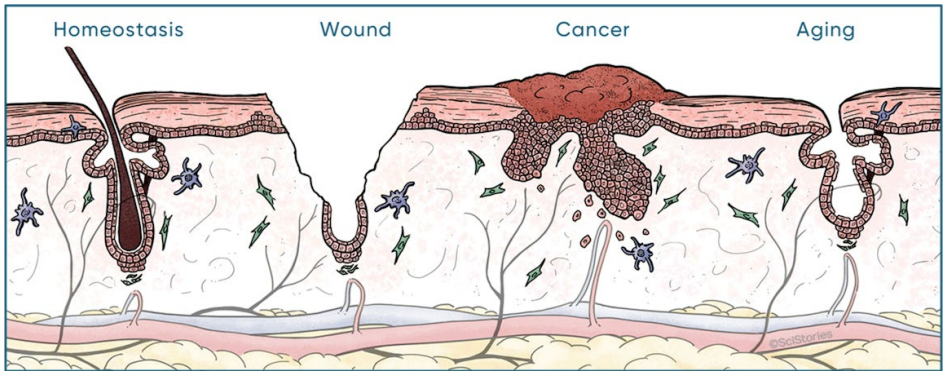
Creativity
Science is fun. Think outside the box.

Rigor
Never underestimate the importance of experimental rigor. It will take you far.

Responsibility
Be a good lab citizen. Do care.

Perseverance
Be faithful to your passion. Be tough.

Freedom
You are here only because you want to be.



NATIONAL INSTITUTES OF HEALTH
THE UNIVERSITY OF TEXAS MD Anderson Cancer Center Making Cancer History®
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DEPARTMENT OF DEFENSE
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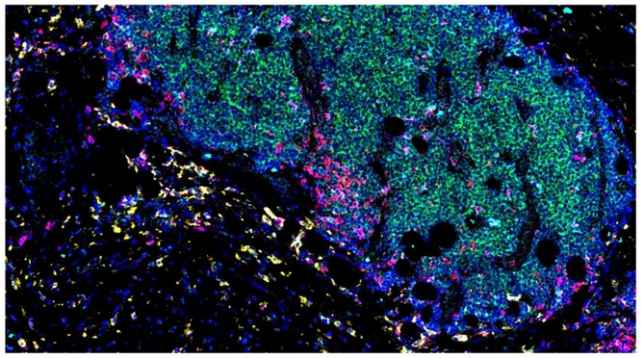
WOMEN & SCIENCE
ROBERTSON FOUNDATION

PI: Jlenia Guarnerio, Ph.D.

Assistant Professor, Department of Genetics
UT MD Anderson Cancer Center.
Contact: JGuarnerio@mdanderson.org
Website: https://www.mdanderson.org/research/departments-labs-institutes/labs/guarnerio-laboratory.html

How does the tumor microenvironment shape cancer progression and therapy response?
Cancer cells do not grow in isolation. They constantly communicate with the surrounding "normal" cells—including immune cells, fibroblasts, endothelial cells, and mesothelial cells—that collectively form the tumor microenvironment (TME)**.** These interactions determine whether tumors grow, evade the immune system, spread to distant organs, or respond to therapy. Our laboratory seeks to understand the molecular mechanisms governing tumor–host interactions and to identify new therapeutic strategies.



Our Research

One of our major research interests is **understanding how cancer-associated fibroblasts (CAFs) suppress anti-tumor immunity.** Using single-cell RNA sequencing, spatial transcriptomics, and genetically engineered mouse models, we discovered that a specialized population of glycolytic CAFs (glyCAFs) accumulates at the tumor margins, where they prevent cytotoxic T cells from entering the tumor through the CXCL16–CXCR6 signaling axis. Disrupting this pathway enhances T-cell infiltration and significantly improves responses to chemotherapy and immunotherapy. Our current work aims to understand how these fibroblasts arise, how their metabolism shapes immune suppression, and how they can be therapeutically reprogrammed to promote effective anti-tumor immunity.
A second focus of the laboratory is understanding **how stromal cells promote metastatic spread**. We recently identified a population of mesothelial-derived CAFs (meso-CAFs) that creates a permissive environment for tumor dissemination within the peritoneal cavity and possibly metastatic colonization of distant organs, including the lung. Using immunocompetent tumor mouse models, lineage tracing, and longitudinal analyses, we investigate how primary tumors systemically reprogram stromal cells to establish metastatic niches.

Our Approach

Our lab combines computational and experimental approaches, including Single-cell RNA sequencing, Spatial transcriptomics, Flow cytometry and high-dimensional immune profiling, Mouse models of cancer, CRISPR-based genetic engineering, Histology and advanced imaging, Molecular and cellular biology.
Students in the lab gain interdisciplinary training spanning cancer biology, immunology, genomics, bioinformatics, and translational research.

Join the Guarnerio Lab!

The Guarnerio Lab was established in 2018 in Los Angeles and relocated to The University of Texas MD Anderson Cancer Center in 2024. We are a collaborative and supportive team of PhD students, postdoctoral fellows, research scientists, and staff who are passionate about understanding the tumor microenvironment and developing innovative cancer therapies.
If you are excited about cancer biology and immunology, we would love to hear from you. ***Visit our lab website or contact us to learn more about research opportunities.***

Wenbo Li Lab (epigenome, 4D Nucleome, enhancers, enhancer RNAs)

--- Seeking PhD students.

A lab picture taken in a recent annual lab BBQ party in Herman Park. Lab members and friends.

Research Summary: The Li lab studies RNA-mediated gene regulation and 3D chromatin organization. We aim to decipher the functions of noncoding DNA and RNA elements in the human genome in gene control and human diseases. We utilize biochemical and -omics approaches (e.g., ChIP-seq, Cut&Tag, Hi-C, PRO-seq, etc.), as well as (epi)genome editing tools and screening (CRISPR/Cas9/dCas9/Cas13). We have about ~60% wet lab components, and ~30-40% bioinformatic components. Students are encouraged to read the previous and recent publications of Dr. Li's lab (**Nature Rev. Genet**. 2016; **Nat. Commun.** 2019, 2024, 2025; **Mol Cell** 2021, **Nature**, 2021, **Cell Research** 2021; **Cell Reports**, 2022; **Nature Cell Biology**, 2022, **Nature Microbiology**, 2023; **eLife** 2024). Full publication list can be found in NCBI MyBibliography: https://www.ncbi.nlm.nih.gov/myncbi/1Jip8J4DFUsQe/bibliography/public/.

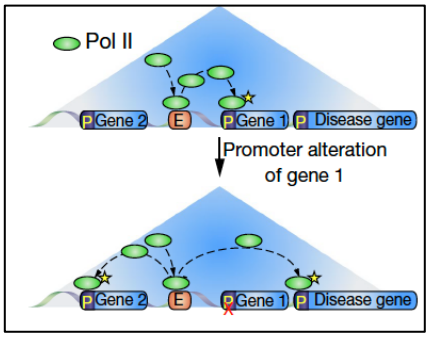


Figure 1*. A diagram showing cancer mutations deregulate cancer genes via Enhancer release and retargeting (ERR).*

Here are some examples of our recent findings. We found that cancer genetic mutations or common human genetic variants can rewire disease gene expression via a novel mechanism called Enhancer release and retargeting (Oh et al., 2021, ***Nature***, *Figure 1*). A set of studies from our lab found that RNA m6A methylation on retrotransposon RNAs can play important roles in deregulating disease-associated "long" genes (Xiong et al., 2021, ***Cell Research***), or that enhancer RNAs (eRNAs) may facilitate gene activation via transcriptional condensates (Lee et al., 2021, ***Molecular Cell***, *Figure 2*). We found that RNA viruses such as SARS-CoV-2 impact host chromatin architecture to deregulate gene expression, which may underlie COVID-19 pathology (Wang et al., 2023, **Nature Microbiology**).

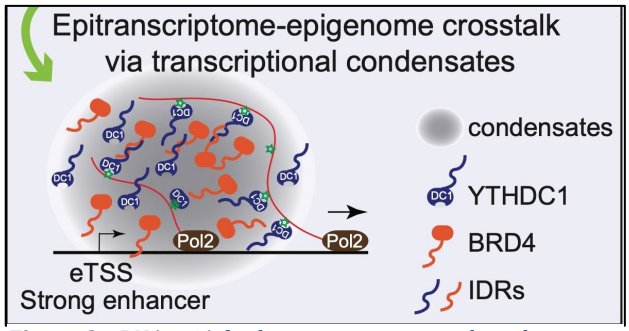


Figure 2*. eRNA m6A facilitates transcriptional condensates.*

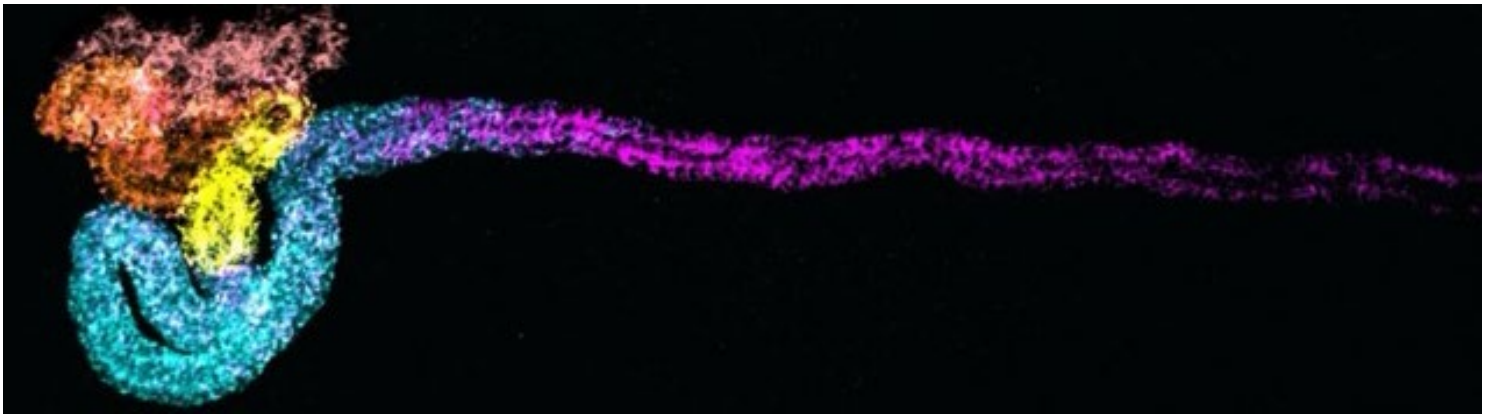
About the lab: We currently have two research faculty Professors, several postdocs, five GSBS PhD students, and two lab assistants (by August 2026). We welcome students with an enthusiasm to uncover fundamental biology and mechanisms of noncoding RNAs, epigenetics, and 3D genome folding, and to pioneer the frontier of RNA medicine. Both experimental and computational approaches are used. Our lab has two locations, one at McGovern Medical School and one at the TMC3 collaborative building (Helix Park). The combination of basic and translational research topics is a strength of our lab.

Our lab intends to take one new PhD student for long-term thesis projects in 2026. One main project is to study enhancer-derived noncoding RNAs in human gene regulation and diseases such as cancer, and to explore novel RNA-targeting therapy for breast cancer and GBM. Another major area we study is related to human neurodevelopment disorders or neurodegeneration, such as Alzheimer's Disease. A potential rotation project is to conduct ChIP-seq (or cut&tag) to examine epigenomic landscapes in breast cancer drug resistance models and learn to conduct some bioinformatic data analysis.

Contact: Wenbo Li, Ph.D., Biochemistry and Molecular Biology, UTHealth McGovern Medical School, and TMC3 collaborative building at Helix Park. Email: Wenbo.li@uth.tmc.edu. You are welcome to inquire via email if you have questions or want to arrange a meeting.

Rachel Miller's Laboratory

How Do Cells Build a Functional Kidney?



How do cells build functional organs?
Our laboratory investigates how developmental signaling, cell adhesion, and cytoskeletal dynamics coordinate kidney development. Using *Xenopus* embryos, CRISPR genome editing, live-cell imaging, and single-cell genomics, we seek to understand how epithelial tissues form during development and how disruption of these processes contributes to congenital disease and cancer predisposition.

Questions We're Answering	Research Opportunity
🧬 How does TP53 build a kidney?	Developmental functions of TP53
🔬 How does β-catenin balance signaling and adhesion?	Live imaging of endogenous β-catenin
🐸 How do developing organs determine cell number?	Organ scaling in polyploid *Xenopus*
🧫 How conserved is kidney development?	Single-cell & spatial genomics

Why rotate with us?
Learn: CRISPR • Live imaging • *Xenopus* • Organoids • Single-cell genomics
Gain: Collaborative lab environment • Supportive mentorship • Scientific independence • Fellowship support • National & international presentations
Succeed: NIH HAI-KUH & CCTS Fellowships • Presidents' Research Excellence Award • Dean's Research Award • Competitive GSBS Scholarships
Alumni Now At: UTHealth • Baylor • University of Pittsburgh • Novartis • AstraZeneca

🧬 **Interested in our lab?** Please reach out to Rachel at Rachel.K.Miller@uth.tmc.edu 🧬

PI: Rodrigo Romero, Ph.D.

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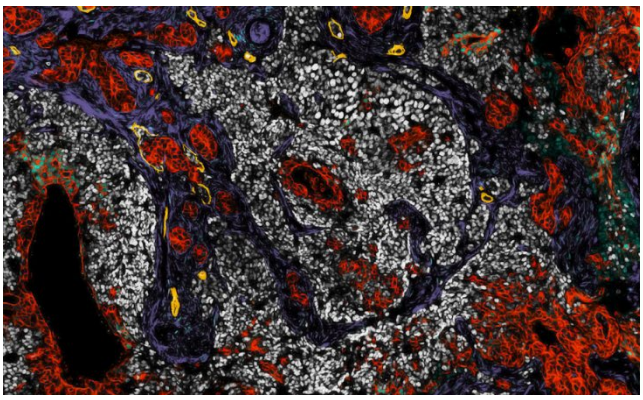
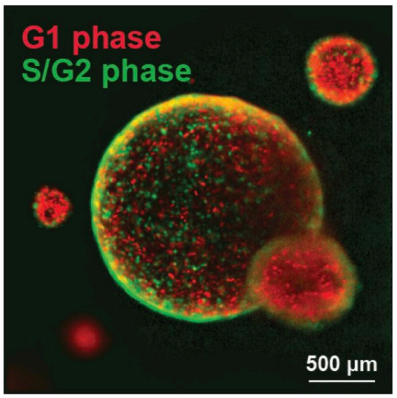
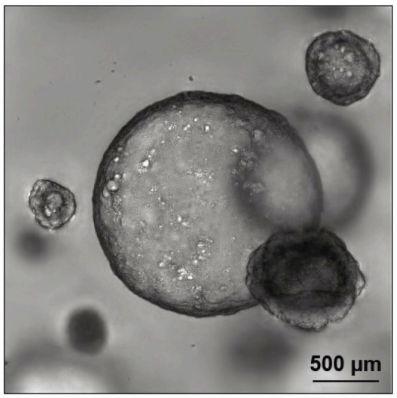






Dissecting how RB1 interaction domains restrict lineage plasticity in cancer

Our overall research goal is to understand the drivers of lineage fidelity during cancer progression. Loss of the tumor suppressor *RB1* is commonly mutated in therapy resistant tumors that progress towards neuroendocrine lineage identities. A paradigm of this state change is observed during the transition from prostate adenocarcinoma to lethal, treatment-resistant neuroendocrine prostate cancer (NEPC). How RB1 mediates cell fate restriction is unclear. RB1 acts a hub with over 300 reported interacting proteins that mediate cell cycle inhibition through the E2F pocket, and organization of repressive chromatin through separable surfaces: an E2F-selective contact linked to EZH2/H3K27me3 silencing, an LxCxE cleft that docks chromatin remodelers, and a C-terminal E2F–DP interface. Which of these functions enforces prostate luminal identity, and which, when lost, unlocks the neuroendocrine state is unknown. This is particularly important given that all described interacting surfaces are lost during prostate cancer progression upon complete loss of the RB1 locus. Our lab uses genetically defined mouse prostate organoids to model this transition (Romero et al., *Nature Cancer* 2024).

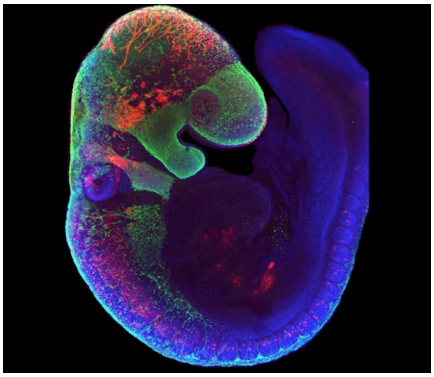
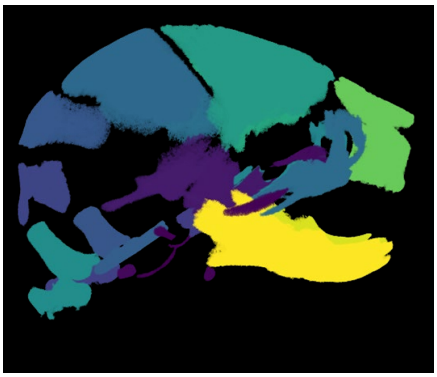
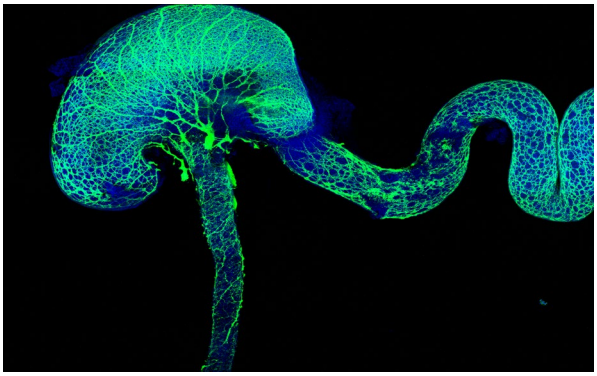
The rotation project: The student will help install and validate a panel of separation-of-function *Rb1* point mutants at the endogenous *Rb1* locus by **prime editing** and ultimately test which mutants fail to restrict neuroendocrine plasticity. The student will use advanced machine learning tools to design and test high-efficiency pegRNAs to prime-edit the murine *Rb1* locus. Depending on interests and timing, a rotation student will take one of three entry points: (1) design, clone, and nucleofect prime-editing reagents into cell lines and organoids, clonally derive lines, and compare editing efficiency through high throughput sequencing reactions, (2) molecular/biochemical: build and validate the parallel epitope-tagged cDNA constructs (including a modular restriction-swap cassette) for chromatin and interactome pulldowns that test whether each mutant rewires RB1's binding landscape; or (3) deploy lineage reporter lines to score how each allele shifts cells along the luminal-to-NEPC axis.

Environment: We are a collaborative lab that pairs mouse cancer genetics with genome engineering and single-cell genomics. Rotation students work closely with a senior mentor on a scoped piece of an active project, are expected to present in lab meeting, and are supported in developing the rotation into a thesis project and fellowship application.

Applying: Please reach out if you are interested in cancer genetics, chromatin biology, or genome engineering! The Romero lab is open for rotation students.

PI: Paul A Trainor, Ph.D.

Vice-Chair, Research, Department of Pediatrics
Chief, Pediatric Research Center
McGovern Medical School
Contact: paul.trainor@uth.tmc.edu
Webpage: https://research.stowers.org/trainorlab/

"Modelling Global Process and Their Tissue Specific Effects in Development and Disease"

Research Focus. A rare disease is defined as having a frequency affecting fewer than 1 in 2000 individuals. Currently over 10,000 rare diseases, which affect more than 300 million individuals world wide have been described, and most of these conditions arise during embryogenesis and early childhood. Of the 1% of individuals that are born with a developmental anomaly, over one-third exhibit facial differences. The Trainor lab therefore studies neural crest cells, which generate most of the bone, cartilage and connective tissue in the head and face, and neurons and glia in the peripheral nervous system, and their roles in development, evolution and disease, with a particular emphais on craniofacial and gastrointestinal development. However, the same genetic, cellular, and biochemical mechanisms that regulate normal vertebrate development and evolution often go awry in the pathogenesis of birth defects. Recently, we have discovered that global processes such as rRNA transcription, ribosome biogenesis, RNA splicing, DNA damage repair, vitamin A metabolism and retinoic acid signaling function in a cell and tissue specific manner during development and in the pathogenesis of disease (Falcon et al PNAS 2022; Moore PNAS 2025; Butler Tjaden 2026; Moore et al Nat Comms 2026; Poverannaya et al Nat Comms 2026; Wiesbeck et al Science 2026). Our overall goal therefore is to define the mechanisms that regulate neural crest cell specification, epithelial to mesenchymal transformation, migration, proliferation, survival and differentiation, and to apply this foundational knowledge to better understand vertebrate development and evolution, and to design theraeputic approaches for prevent rare congenital disorders.

Environment. My laboratory is highly collaborative with a reputuation for collegiality, a strong dedication to education and training, and a long standing commitment to individual development training plans and mentored trainee success.

Applying. My lab is new to UTHealth and growing! Please get in touch if you are interested in discovering fundamental principles underlying development, evolution and the pathogenesis of disease, as well as designing therapeutic approaches for preventing congenital pediatric diosrders.

Marianna Trakala, Ph.D.
Assistant Professor & CPRIT Scholar in Cancer
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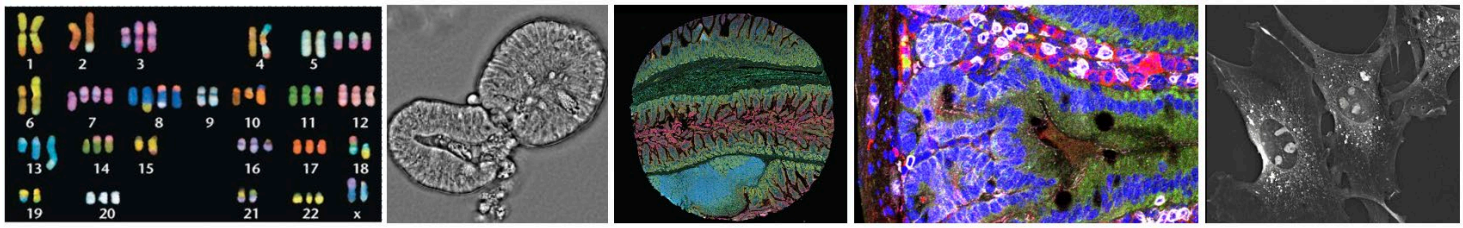
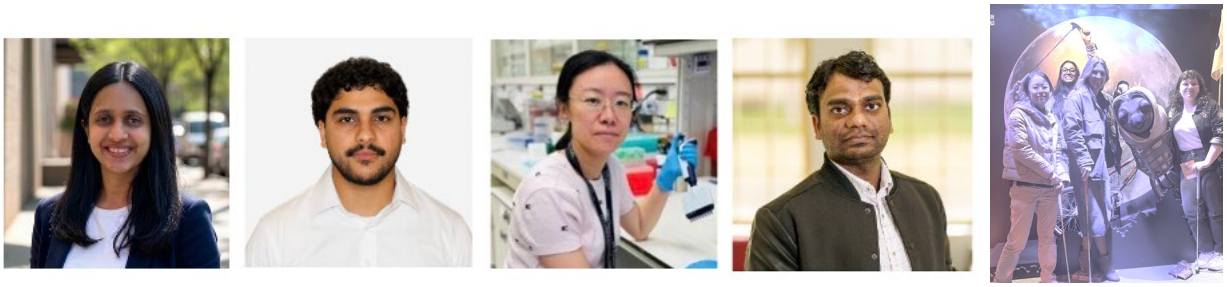
https://faculty.mdanderson.org/profiles/marianna_trakala.html



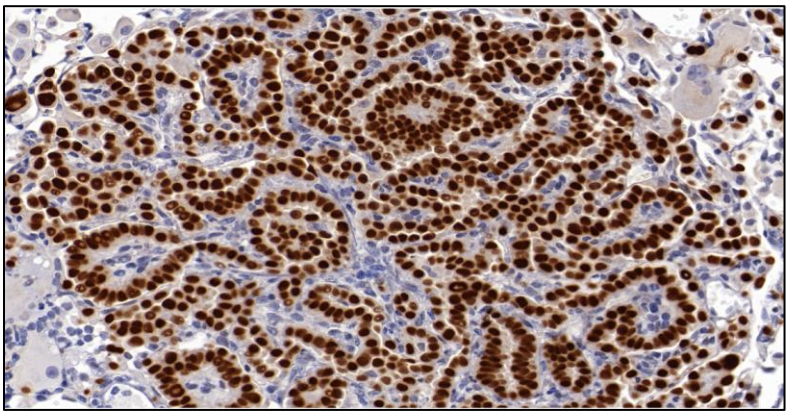
Chromosomal instability, aneuploidy, and immune surveillance in cancer

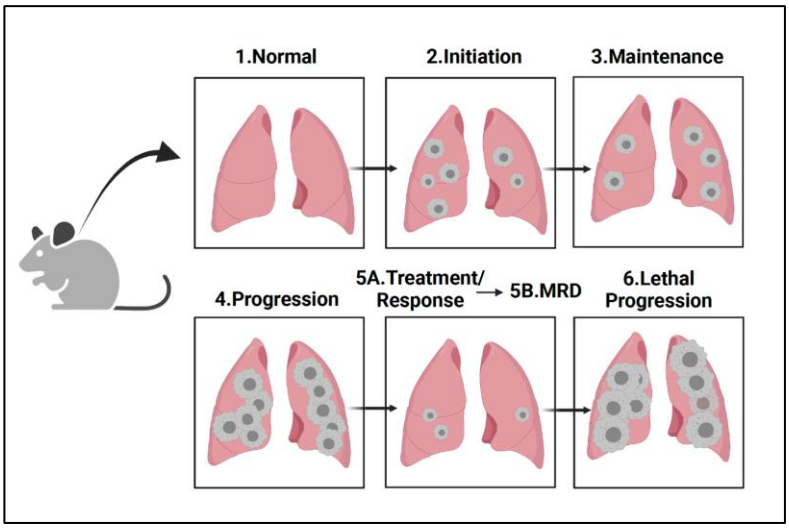
Our research focuses on understanding the consequences of chromosomal instability and aneuploidy in vivo. Both are defining features of many cancers, yet their effects on tumor development, tissue physiology, and immune recognition remain incompletely understood. We are particularly interested in defining the molecular consequences of aneuploidy and determining how these alterations influence interactions between aneuploid cells and the immune system. Our goal is to understand how immune cells sense and potentially limit the persistence of aneuploid cells in vivo, as well as how highly aneuploid tumors develop mechanisms to evade immune surveillance. By identifying pathways that determine whether aneuploid cells are eliminated or protected, we aim to uncover vulnerabilities that could ultimately be exploited to improve immune-based therapies for highly aneuploid cancers.

Rotation Projects: 1) Effect of aneuploidy on antigen-specific T-cell killing. Cancer cell lines expressing ovalbumin will be induced to missegregate chromosomes, generating aneuploid cells that can be compared with euploid controls. Using OT-I T cells that specifically recognize the SIINFEKL peptide, the student will evaluate how aneuploidy affects antigen presentation, immunogenicity, and T-cell-mediated killing, including after perturbation of selected components of the antigen-processing and presentation pathway. 2) Validation of intrinsic regulators of NK-cell recognition of aneuploid cells. Our preliminary studies have identified cell-intrinsic transcriptional programs induced by chromosome missegregation that influence the recognition and killing of aneuploid cells by NK cells. The student will perturb selected candidate genes through knockdown or overexpression and assess their effects on NK-cell-mediated killing to validate these findings and begin defining the underlying molecular mechanisms.



Environment: We think boldly, support one another and have fun while we are making exciting discoveries along the way. **Apply:** Please contact me if you are interested in training in our group.





Hello Potential PhD Students!

PI: Aria Vaishnavi, Ph.D.

Assistant Professor
Department of Genetics
UT MD Anderson Cancer Center,
Contact: avaishnavi1@mdanderson.org

Research in the Vaishnavi lab uses the mouse lung (depicted in the cartoon to the left) as a model, and applies mouse genetics, environmental exposures, and biochemical techniques to dissect the molecular mechanisms underlying lung injury and wound repair. We study how the dysregulation of these events leads to human diseases, including pulmonary fibrosis, as well as cancer.

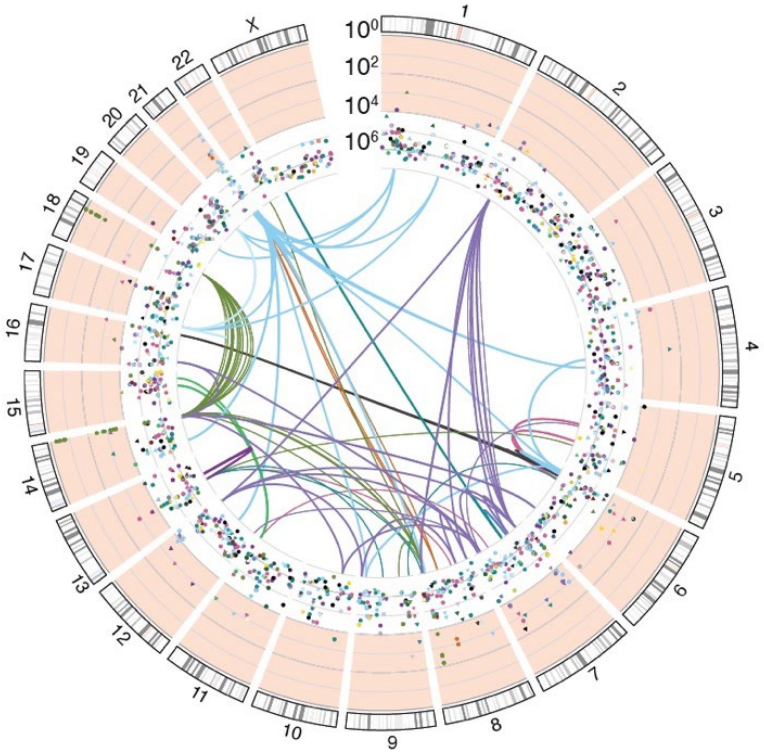
As tobacco usage continues to decease, the proportion of never smoker lung cancer is rapidly increasing (risk depicted in the cartoon below). Using state-of-the-art mouse models and technology, we have recently created novel exposure mouse models to help us understand how air pollution and radon gas cause lung cancer. These topics include several potential rotation project options to choose from, ranging from studying the role of tumor cell instrinsic signaling, or tumor cell extrinsic mechanisms like inflammation or cancer associated fibroblasts, metabolism, and the microrbiome.

We are also excited to report our investigation on the role of RTK ligands and autocrine signaling in supporting oncogene-driven lung cancer has just been accepted (Dacheux MA et al, *in-press*). This study has strong implications on the role of autocrine support signaling on lung cancer initiation, progression and therapeutics. We are now investigating the role(s) of such support signaling pathways in promoting minimal residual disease (MRD) and how that drives acquired resistance in different models of oncogene-driven lung cancer. This will be another rotation project option you can choose from.

Please check out our website: https://www.mdanderson.org/research/departments-labs-institutes/labs/vaishnavi-laboratory.html
We are ALWAYS excited to recruit enthusiastic scientists to join our team!

CANCER PREVENTION & RESEARCH INSTITUTE OF TEXAS
THE UNIVERSITY OF TEXAS MD Anderson Cancer Center
HUNTSMAN CANCER INSTITUTE UNIVERSITY OF UTAH
LUNG CANCER RESEARCH FOUNDATION
NIH NATIONAL CANCER INSTITUTE

Cancer Genomics and Evolution Laboratory – Prof. Peter Van Loo



Our research focuses on large-scale pan-cancer genomics to gain insight into the genes, mutational processes, and evolution of cancer. Our work is highly data-driven, with a focus on large-scale data analysis to gain broad biological insight, and on the development of computational methods to enable conceptually novel analyses. Our research group is mostly computational with a small wet-lab component.

The cancer genome contains an archeological record of its past. The Cancer Genomics and Evolution Laboratory has pioneered methods to reconstruct a cancer's life history from massively parallel sequencing data and uses these 'molecular archeology of cancer' approaches to obtain detailed timelines of tumor evolution across many cancer types.

Since its inception, members of the Cancer Genomics laboratory have co-authored 18 papers in Nature, Science or Cell. Recent successes include pan-cancer studies of the evolutionary history of cancer (**Gerstung *et al.*, *Nature* 2020**, **Baker *et al.*, *Cancer Discovery* 2024**), intra-tumor heterogeneity (**Dentro *et al.*, *Cell* 2021**), the mutational landscape in non-unique regions of the human genome (**Tarabichi *et al.*, *Nature Biotechnology* 2021**), and biallelic mutations (**Demeulemeester *et al.*, *Nature Genetics* 2022**).

Environment

We are a bold, imaginative, open, dynamic and collegial team. Students are mentored to complete ground-breaking research projects that helps us understand how cancers evolve.

Rotation projects

Our lab is growing, funded by NIH, CPRIT, and DoD. This year, we offer rotation projects in computational cancer genomics, including (i) studying evolution of extrachromosomal DNA in cancer using single-cell DNA sequencing; (ii) using circulating tumor DNA to track tumor evolution over treatment; and (iii) developing new computational approaches to integrate spatial and single-cell data in cancer to understand how tumors develop and grow.

Please contact me at pvanloo@mdanderson.org if you are interested in working with us!

More info

https://www.mdanderson.org/research/departments-labs-institutes/labs/van-loo-laboratory.html

PI: Jun Wang, PhD

Professor
Department of Pediatrics, McGovern Medical School
The University of Texas Health Science Center at Houston
Email: jun.wang@uth.tmc.edu
Website: https://med.uth.edu/pediatrics/faculty/jun-wang-ph-d/

Wang lab studies signaling pathways such as Hippo, Wnt and Bmp pathways as well as non-coding RNAs in regulating craniofacial and cardiovascular development, diseases and regeneration, using approaches include a combination of genetic mouse models, cell models, molecular/biochemical techniques, electrophysiology techniques, imaging, cell culture/manipulation, CRISPR-Cas9 genome editing, and cutting edge next generation sequencing techniques such as single cell multiomics (scRNA-seq and scATAC-seq) and Cut&Run/Cut&Tag seq.

Projects in the Wang lab focus on: **1) Neural Crest Cells (NCCs)**, stem cells that make significant contributions to different tissues/organs, including the heart and head, and defects in NCCs give rise to many diseases. Projects study NCCs proliferation/migration/stemness/cell fate decisions and NCCs derived heart development and congenital heart diseases, as well as NCCs derived cranial skeleton formation, repair and regeneration. **2) Cardiac Conduction System (CCS)**, the tissue network in heart initiates and maintains normal heart contractions. Projects study CCS development, homeostasis, and regeneration, as well as CCS aging and diseases.

Wang Lab Environment. Wang lab is a highly collaborative team, consisting of postdoctoral fellows, graduate students, research associates, research assistants, and undergraduate researchers. The PI has been devoted to mentoring trainees and helping them to reach their career goals. Trainees actively attend national or international meetings and have received numerous honors and awards, including AHA predoctoral and postdoctoral fellowships, NIH F31 and K99 awards. Students will also take advantage of both in-lab collaborations and active collaborations with other labs, including local, national, and international collaborations. Please feel free to ask our current PhD students about the Wang lab: **Mark Returan**, **Julianna Quinn** and **Shannon Erhart**.

PI: Wenyi Wang, Ph.D.
Professor
Department of Bioinformatics and Computational Biology
The University of Texas MD Anderson Cancer Center
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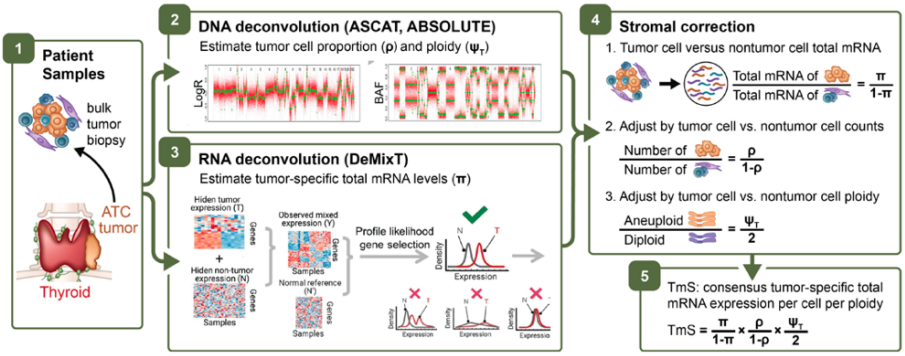
THE UNIVERSITY OF TEXAS MD Anderson Cancer Center
Making Cancer History®

Cancer genomics and statistical bioinformatics lab

The Wang Lab develops computational methods to unravel the complexity of cancer, focusing on tumor evolution, cell-type-specific transcriptional activity, and clonal architecture. Our current research centers on multi-omic deconvolution to study DNA–RNA dynamics and cancer risk modeling using machine learning and Bayesian approaches. Collaborating closely with clinicians and experimental biologists, we translate data-driven insights into clinically meaningful advances. We are equally committed to building a research environment where statistical rigor and artificial intelligence drive innovation in cancer discovery.



We have made pioneering contributions to developing methods and software tools for tumor heterogeneity and evolution, including MuSE for subclonal mutation calling, with MuSE2.0 offering a 50-fold speed increase (***Ji S, et al., Genome Res. 2024***); and a pan-cancer characterization of intra-tumor heterogeneity in subclonal selection (***Dentro S, et al., Cell 2021***). In cancer transcriptomics, we developed DeMixT for cancer-cell specific deconvolution (***Wang Z, et al., iScience 2018***), and further developed a method to quantify *tumor-specific total mRNA expression* (*TmS*) at scale from matched RNA/DNA sequencing data, which is demonstrated to be a potential pan-cancer biomarker of prognosis and treatment response (***Cao S, et al., Nat Biotechnol 2022,*** see Illustration above). Building on TmS, we recently showed that it stratifies chemotherapy response in triple-negative breast cancer across multi-ethnic cohorts, outperforming established molecular subtypes (***Dai Y, et al., Cell Rep Med 2026***). We also introduced DeMixSC for estimating cell type proportions in complex tissues (***Guo S, et al., Genome Res. 2025***), DeMixNB for deconvolving sparse-count RNA sequencing data (***Montierth MD, et al., Genome Biol, accepted***), and a guide to over 40 deconvolution methods (***Dai Y, et al., Nat Rev Cancer 2026***).

Our cancer risk prediction modeling has been focused on hereditary cancer syndromes associated with germline *TP53* mutations. We have developed models to predict the risk of *de novo* germline mutations and the risk of multiple unique primaries (***Nguyen NH, et al., Ann Appl Stat 2025***) or cancer-specific risks in individual patients, as well as tools to be used by clinicians and genetic counselors to provide quantifiable cancer risks to help guide clinical decisions (***Nguyen NH, et al., J Clin Oncol 2024, JCO CCI 2024***). In a prospective clinical cohort, our models predicted TP53 carrier status more accurately than current guidelines (***Corredor JL, et al., Am J Hum Genet 2026***). Currently, we are investigating the unique contributions of different *TP53* mutations on cancer development and how to stratify patients' risk based on these differences. Our original research has also been published in prestigious medical/biological journals and top statistics journals, e.g., *Cancer Research* and *JASA.*

Environment Our group thrives in a collaborative environment, with strong partnerships across clinical cancer genetics and breast, colorectal, prostate and thyroid cancer research. Many former trainees continue to collaborate with the lab in their academic or industry roles, fostering a lasting and synergistic network.

Join us Rotation projects range from developing deconvolution and deep-learning methods for cell-type-specific activity to analyzing spatial and single-cell data across several cancer types, integrating them with genomic and proteomic data to uncover clinically relevant signals. We welcome enthusiastic PhD or master's students excited to jump in and explore these questions with us.

For more information on current projects in the lab please visit the lab website.

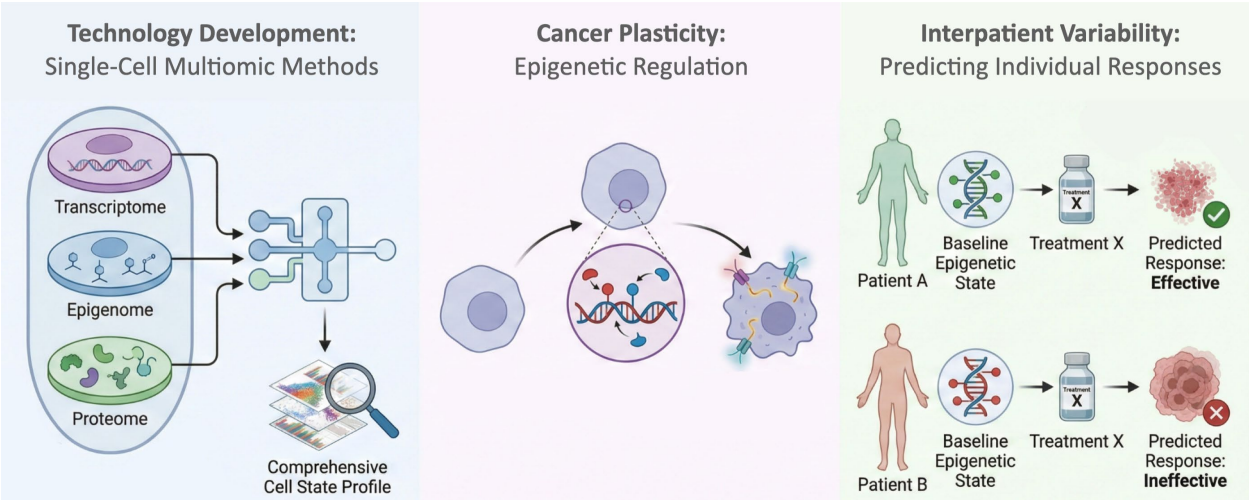
BINGJIE ZHANG LABORATORY

Single-cell multimodal omics to understand cancer plasticity and treatment response

Bingjie Zhang, Ph.D.
Assistant Professor, Department of Systems Biology
Email: bzhang12@mdanderson.org

ABOUT THE LAB

The Zhang Lab develops and applies advanced single-cell genomic technologies to uncover the molecular mechanisms that affect tumor behavior. We seek to measure multiple layers of cell state, including the transcriptome, epigenome, proteome and beyond, within the same cell, then integrate these data to reveal states that may be invisible to conventional assays. Our research connects three goals: inventing sensitive multimodal methods; defining epigenetic mechanisms of cancer plasticity as cells adapt, evolve and resist therapy; and identifying baseline epigenetic features that may explain interpatient variability and predict treatment response.



Research directions: multimodal technology development, cancer plasticity and interpatient variability in therapeutic response.

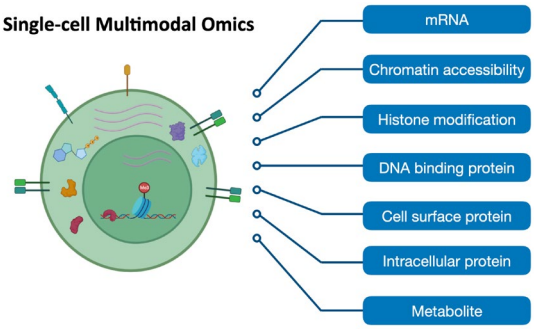
TRAINING ENVIRONMENT

Established in 2025, the lab is a small, growing team with two postdoctoral fellows and is actively recruiting its first Ph.D. student. Trainees receive close access to the PI, hands-on mentorship and an opportunity to help shape the lab's scientific culture.

POTENTIAL ROTATION PROJECT

Multimodal profiling in a cancer cell line

Project overview. The rotation student will learn the conceptual and experimental foundations of single-cell multiomics by applying a joint-profiling workflow to an established cancer cell line. During the rotation, the student will learn to culture cells, prepare samples, perform a multimodal assay, and generate sequencing libraries. They will also gain experience troubleshooting experiments and evaluating assay quality. After sequencing, they will receive guided, hands-on training in basic FASTQ data processing and quality-control analysis.



Training and fit. The project emphasizes wet-lab experimentation and does not require prior bioinformatics experience. By the end of the rotation, the student will have developed an understanding of multimodal study design and gained hands-on experience conducting and evaluating multimodal experiments. We especially welcome curious, motivated trainees interested in epigenetic regulation in cancer.

Zhang Lab Research

Cellular Homeostasis in Neurodegeneration

IMM, UTHealth Houston • PI: Sheng Zhang, PhD

RESEARCH PROGRAM

Age-related **neurodegenerative disorders** cause progressive neuronal damage, leading to memory loss, movement problems, and serious disability. Dysfunction in cellular homeostasis mechanisms (e.g., autophagy, endolysosomes, chaperones) is the leading cause of these devastating diseases. The Zhang Lab investigates how these cellular protective systems operate and why their failure drives toxic protein buildup and neurodegeneration in long-lived neurons.

CORE SCIENTIFIC QUESTIONS

- Why different neurodegenerative diseases affect different neuronal populations?
- How do cellular homeostasis mechanisms operate to recognize, sort and clear toxic materials while preserving healthy cellular components?
- How does the Hsp110/Hsp70/Hsp40 chaperone machine operate to protect neurons under normal and disease conditions?

APPROACHES AND MODEL SYSTEMS

- Integrated genetics, neuroscience, biochemistry, and cell biology.
- Model systems: *Drosophila* (fruit flies), mammalian cell culture, and mouse models.

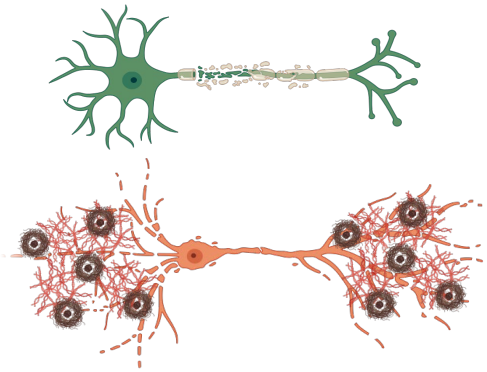
ROTATION PROJECTS

- The functions and regulation of Huntington's disease gene Huntingtin.
- The mechanism that coordinates membrane trafficking network (e.g., autophagic and distinct endolysosomal systems).
- The Hsp110/Hsp70/Hsp40 chaperone mechinery in protein homeostasis and neurodegenerative disorders.

TRAINEE RECORDS:

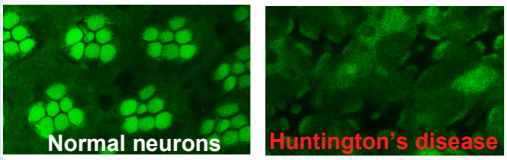
President's Research Excellence Awards, NIH F99-K00 award,
John J. Kopchick Fellow, NIH Outstanding Scholars in Neuroscience Award (OSNAP), and more...

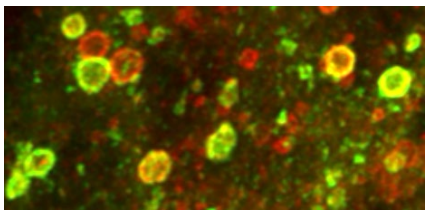
Long-lived neurons rely on robust cellular homeostasis mechanisms for healthy longevity



Dysfunctional cellular homeostasis underlies tangle/plaque formation and neurodegeneration

Central nervous system in fruit fly





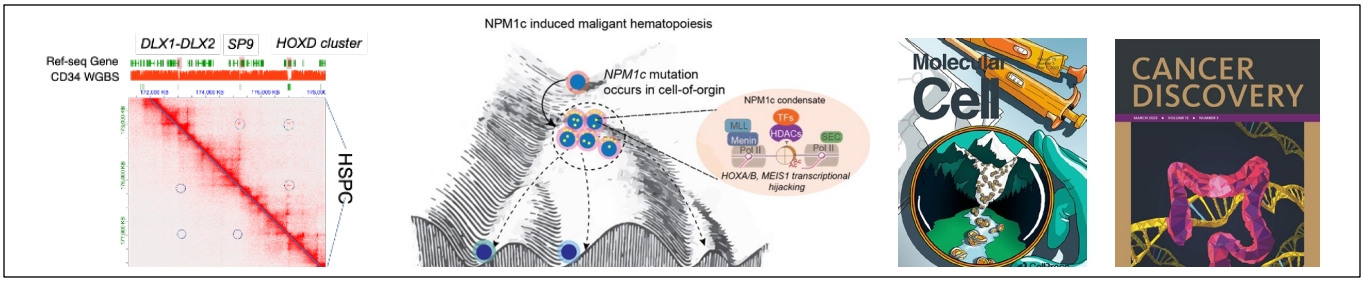
Recycling and Degradative endosomes

PI: Xiaotian Zhang, Ph.D.

Assistant Professor, Department of Biochemistry and Molecular Biology.

UTHealth Houston, McGovern Medical School.

Email: xiaotian.zhang@uth.tmc.edu



Spatial and temporal regulation of gene expression is a key to organismal development. Such precise regulation requires multi-layer of gene expression regulation on transcription, including transcription factor binding, histone modification, DNA modification, and 3D genomic organization. Amazingly, such regulations involve millions of regulatory elements (cis-regulatory elements: enhancers, insulators, etc.) orchestrate on a daily basis to ensure the performance of normal physiological and developmental processes. The dysregulation of such processes could lead to the ectopic gene expression that initiates multiple diseases, particularly cancer.

Focusing on the epigenetic regulation, the Zhang lab produced significant work on the following aspects:

(1). The discovery of extremely long 3D genome interactions between Polycomb target loci in hematopoietic stem cells (Zhang et al, Molecular Cell 2020).

(2) The discovery of the mutant NPM1 protein as a transcriptional amplifier on chromatin, which transformed the study of this common leukemic mutant protein in acute myeloid leukemia (Wang et al, Cancer Discovery, 2022).

(3). The discovery of novel non-coding genomic elements in globin switch regulation (Himadewi, et al, eLife, 2021).

We are currently working on the following research topics:

(1). *The transcriptional hijacking mechanism by mutant NPM1 protein in acute myeloid leukemia (AML) through IDR and liquid-liquid phase separation.*

(2). *The regulation and therapeutic targeting of extreme long-range Polycomb loci interactions in B cell ALL and lymphoma.*

(3). *Develop small molecular inhibitors to remodel 3D genome in the hemoglobin switch and the novel gene therapy for hemoglobinopathies.*

Environment. We have a very welcoming and supportive laboratory culture, appreciating the contributions of each lab member to make innovative discoveries in biomedical research. PI will be actively involved in the mentoring and guidance of junior lab members.

Contact. Please contact Xiaotian Xiaotian.zhang@uth.tmc.edu. Welcome to join our lab for rotation!