# Genetics & Epigenetics Program

**Research Summaries of Faculty Seeking Students**
**Partial List as of 8/14/25**
*See all faculty research profiles on the GSBS website*

## Genetics & Epigenetics Program Faculty Seeking Students

**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 PhD students)

**Yejing Ge, PhD,** Cancer Biology, MDA (accepting PhD students)

**Jlenia Guarnerio, PhD,** Genetics, MDA (accepting PhD students)

**Jayhun Lee, PhD**, Microbiology & Molecular Genetics, UTHealth Houston (accepting PhD students)

**Yonathan Lissanu, MD, PhD**, Thoracic & Cardiovascular Sugergy & Genomic Medicine, MDA (accepting MS & PhD students)

**Yuan-Hung Lo, PhD**, Molecular & Cellular Oncology, MDA (accepting PhD students)

**Guillermina Lozano, PhD**, Genetics, MDA (accepting PhD students)

# Genetics & Epigenetics Program Faculty Seeking Students

**Rachel Miller, PhD,** Pediatrics, UTHealth Houston (accepting PhD students)

**Peter Van Loo, PhD,** Genetics, MDA (accepting PhD students)

**Jun Wang, PhD,** Pediatrics – Research, UTHealth Houston (accepting PhD students)

**Wenyi Wang, PhD,** Bioinformatics & Computational Biology, MDA (accepting MS & PhD students)

**Zhichao Xu, PhD**, Biochemistry and Molecular Biology, UTHealth Houston (accepting PhD students)

**Xiaotian Zhang, PhD**, Biochemistry and Molecular Biology, UTHealth Houston (accepting PhD students)

**Zhongming Zhao, PhD,** School of Biomedical Informatics and SPH, UTHealth Houston (accepting MS & PhD students)

**Ye Zheng, PhD**, Bioinformatics & Computational Biology, MDA (accepting PhD students)

# Genetics & Epigenetics Program Faculty Seeking Students

***Faculty are listed in one or two categories***

## Cancer Genetics:

Andrew Dunbar, MD
George Eisenhoffer, PhD
Yejing Ge, PhD
Jlenia Guarnerio, PhD
Yonathan Lissanu, MD, PhD
Yuan-Hung Lo, PhD
Guillermina Lozano, PhD
Peter Van Loo, PhD
Wenyi Wang, PhD
Zhichao Xu, PhD
Xiaotian Zhang, PhD
Ye Zheng, PhD

## Epigenetics:

Andrew Dunbar, MD
Yejing Ge, PhD
Yonathan Lissanu, MD, PhD
Yuan-Hung Lo, PhD
Peter Van Loo, PhD
Zhichao Xu, PhD
Xiaotian Zhang, PhD
Zhongming Zhao, PhD
Ye Zheng, PhD

## Developmental Genetics:

Swathi Arur, PhD
Richard Behringer, PhD
George Eisenhoffer, PhD
Jayhun Lee, PhD
Rachel Miller, PhD
Jun Wang, PhD

## Human Genetics:

Rachel Miller, PhD
Jun Wang, PhD
Zhongming Zhao, PhD

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

*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

**What do we do?:** We use multidisciplinary approaches and model systems with a goal to gain knowledge into three specific biological questions. We hope to understand the basis of (i) environmental signaling and its role in male and female fertility, (ii) signaling and control of birth defects, with a specific focus on the Ras pathway and (iii) signaling based control of post-transcriptional regulation on cancer metastasis.

Below, I provide highlights of some of our ongoing research.

**I. Nutritional programs that govern female germ cell development and transition to embryo development.** Female meiosis I is completed *in utero* in vertebrates. Defects in meiosis I during female germ cell development manifest as sterility in later in her life, or as birth defects in her children. While we assume that maternal health and nutrition influences progeny health, we just never knew that maternal nutritional status regulates *female child's germ cell health* as well, until our lab discovered a direct link between maternal nutrition and regulation of female meiosis I and oocyte development. Trainee publications: *Lopez and Chen et al., Developmental Cell, 2013; Suen et al., Nat Str Mol Biol, 2013; Mattingly et al., J Biophy, 2015; Das et al., Science Advances 2020; Das et al., PNAS, 2022; Trimmer et al., Cell Reports, 2023, Baek et al., Cell Reports, 2025, Baek et al., Current Opinion in Cell Biology, 2025.*


Lab team, 2023-2024. L-R (front): Jacob Ortega, Nick Newkirk, Swathi Arur, Tokiko Furuta, Melany Puente, Amelia Li, Janet Cheng.

L-R (back): Shin-Yu Chen, Deba Das, Lisa Watson, Kennty Trimmer, Han Bit Baek

Talk to them to learn about the lab culture!

**Current students working on this broad topic**: You?

**II. Small RNA pathways that control development**: We discovered a direct intersection between environmentally activated signaling pathways and production of small RNAs that controls oocyte development, and oocyte to embryo transition. The discoveries include understanding the role of Dicer and Drosha phosphorylation, small RNA production, and determining why subsets of populations of small RNAs are generated, and what this may mean. Trainee publications: *Drake et al., Dev Cell, 2014; Minogue et al., Nat Comm, 2018; Minogue et al., Current Prot, 2019; Aryal et al., 2018, PNAS, Ortega et al., Science Adv, 2024*

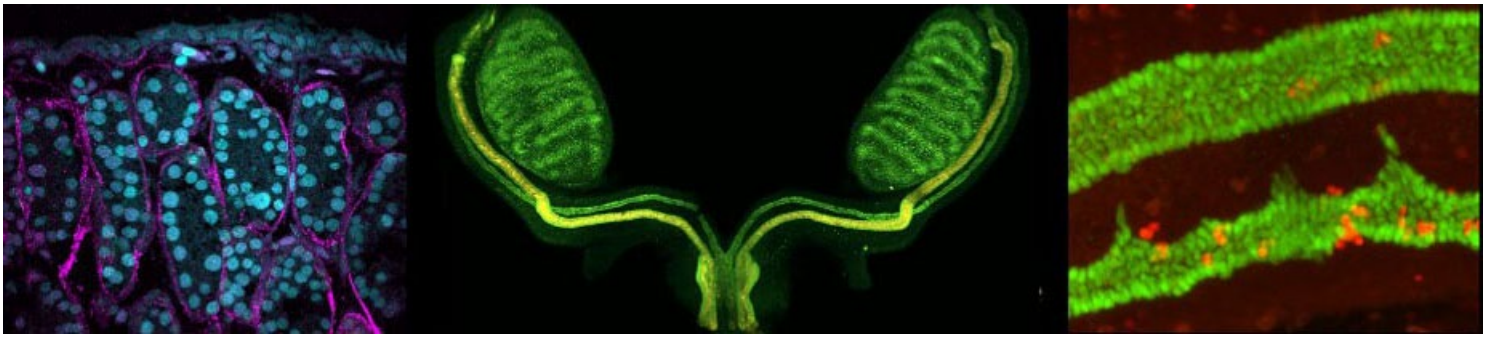
**Current students working on this broad topic**: Nick Newkirk, & You?

**III. Dicer phosphorylation and nuclear role in cancer development**: We discovered that Dicer is phoshorylated and translocated to the nucleus in *C. elegans*. We then generated mouse models to determine its role in cancer development. Phosphorylated nuclear Dicer drives tumor spread in mouse models of oncogenic KRas and mutant p53. We then discovered in non-small cell lung cancers that phosphorylated nuclear Dicer does not regulate microRNAs, instead it forms a large chromatin complex in the nucleus which helps open chromatin and affect transcription of lineage defining genes resulting in lineage reprogramming of lung tumor cells to gastric lineage. Trainee publications: *Aryal et al., 2018, Cancer Res; Reyes et al., 2023, Science Advances.*

**Current students working on this topic**: You?

# Richard Behringer, Ph.D.

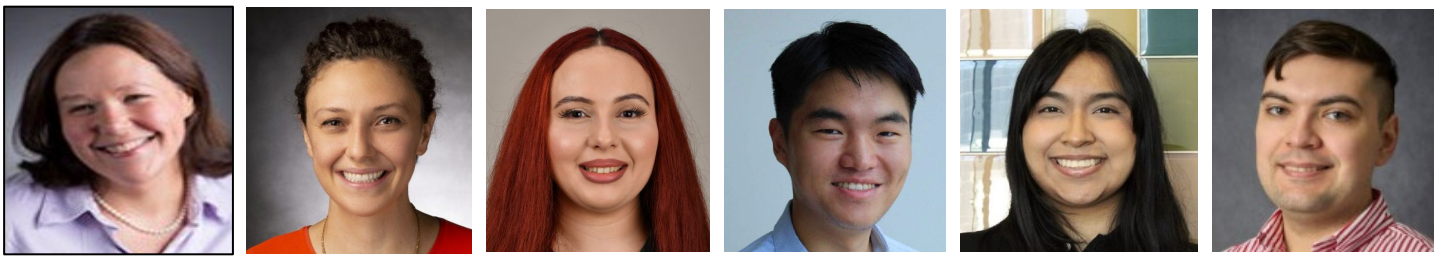
Professor
Department of Genetics
MD Anderson Cancer Center, BSRB S11
rrb@mdanderson.org
https://faculty.mdanderson.org/profiles/richard_behringer.html



## 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 and Diffences in Sex Development. Alterations in reproductive organ formation are associated with infertility, high risk pregnancies, and miscarriage. Our primary model system is the mouse but we also have a colony of brown anole lizards to explore these 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.



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

Dr. Andrew Dunbar, MD, is an Assistant Attending in the Department of Leukemia and Assistant Professor in the Department of Hematologic Biology and Malignancy program at MD Anderson Cancer Center. He is currently the head of a new, translational research laboratory on MD Anderson's South Research Campus, and his research focus includes primarily the study of chronic blood cancers known as **Myeloproliferative Neoplasms** and other myeloid blood cancers. Dr. Dunbar recently joined the MD Anderson faculty in January 2024. Prior, he completed his clinical oncology fellowship and research training in the laboratory of Dr. Ross Levine at Memorial Sloan Kettering Cancer Center in New York, NY. Dr. Levine is an international leader in the study of MPNs and was one of the early investigators to identify JAK2 and MPL mutations in these diseases.

As a leader of his own lab, Dr. Dunbar wishes to identify the processes by which a chronic, smoldering MPN evolves into a more aggressive form of the disease. Patients with advanced MPN often present with severe bone marrow scarring (i.e. fibrosis), yet researchers still do not fully understand the biological processes that contribute to this. Using cutting-edge, "multi-modal" next-generation sequencing techniques, he wishes to uncover how abnormal MPN blood cells communicate with one another and surrounding normal bone marrow cells to force cells to produce scar tissue. Moreover, he is interested in learning how DNA mutations in MPN cells cooperate with one another to promote disease progression. For example, while many MPN patients have mutations in the JAK2 gene, it is also not uncommon to find other "high-risk" mutations such as ASXL1, EZH2 that, when present, predict poor response to treatments and progression to leukemia. Dr. Dunbar wants to understand the biological mechanisms as to why this is the case so that he can identify new therapies for these high-risk patients.

Dr. Dunbar is also interested in finding the next-generation of treatments for MPNs. Currently he is working with his mentor, Dr. Levine, in developing a whole new class of "second generation" JAK inhibitors that are much more potent and selective than the current agents. The goal with these drugs will be to enhance the effect of JAK inhibition so that MPN cells can be more effectively irradicated. Excitingly, these "second generation" JAK inhibitors are now in early-phase clinical trials, so Dr. Dunbar will have the opportunity to evaluate—real time—how patients on trial respond to these drugs. Finally, he hopes to explore how other promising investigational agents currently in MPN clinical trials, including BET inhibitors, XPO1 inhibitors, etc. work to fight against MPN cells. Ultimately, the goal will be to look at the differences between those who respond to the drugs versus those who do not. If he can identify "biomarkers" that better predict the likelihood of response to a particular therapy, doctors may be in a better position to tailor specific therapies for individual patients.

Although Dr. Dunbar's lab is relatively new, he has already established important relationships with other MDACC researchers, including our MPN clinicians Drs. Prithviraj Bose, Lucia Masarova, and Pemmaraju, to discuss potential opportunities for collaboration. Ultimately, he is eager to further expand the MPN translational research program to create a large, MPN research hub built off the already-powerful MPN clinical trials program that exists at MD Anderson.

THE UNIVERSITY OF TEXAS
MD Anderson Cancer Center


# George T. Eisenhoffer, PhD

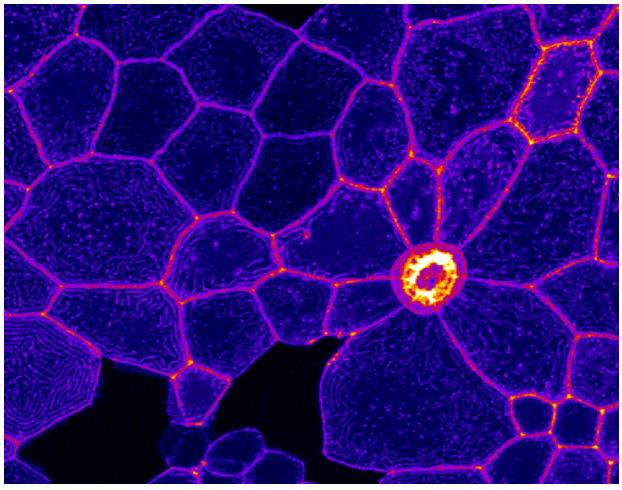
Associate Professor
Department of Genetics
gteisenhoffer@mdanderson.org

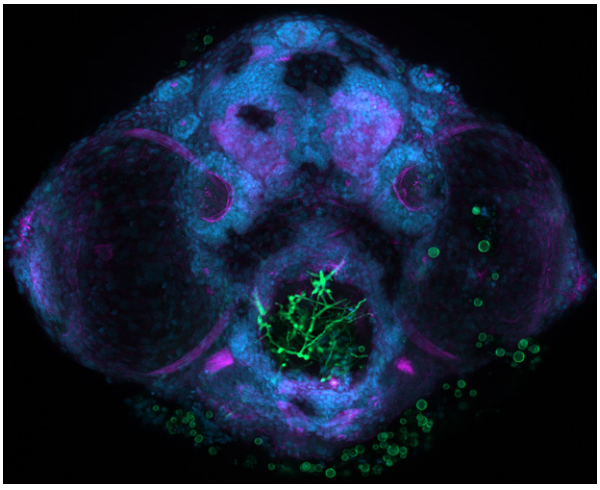

## Research Interests

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 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). 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). 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.



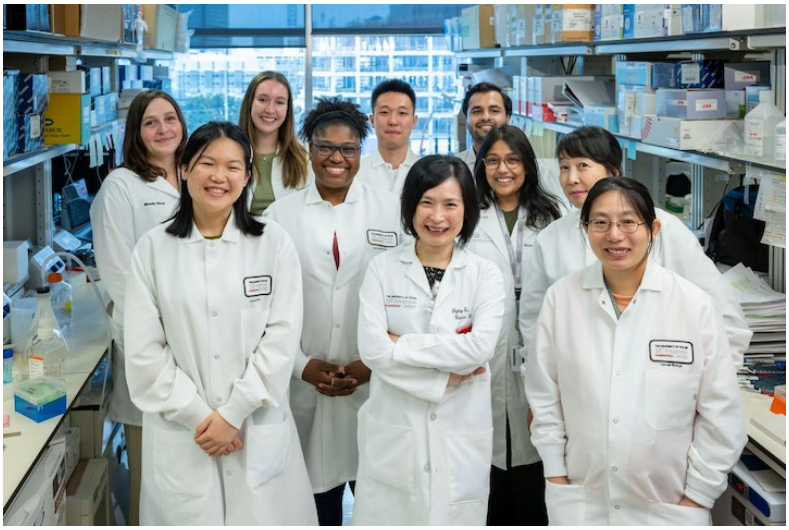


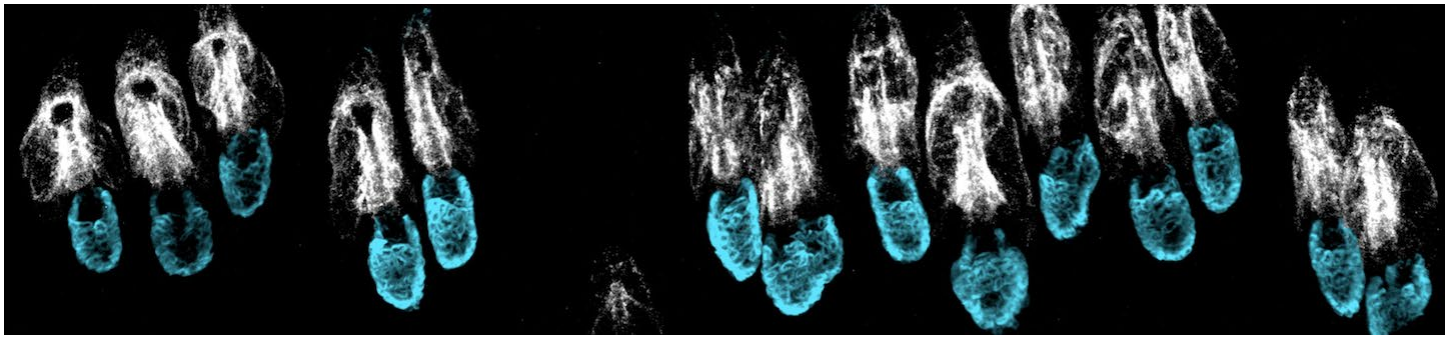
***Seeking PhD Student***

## 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

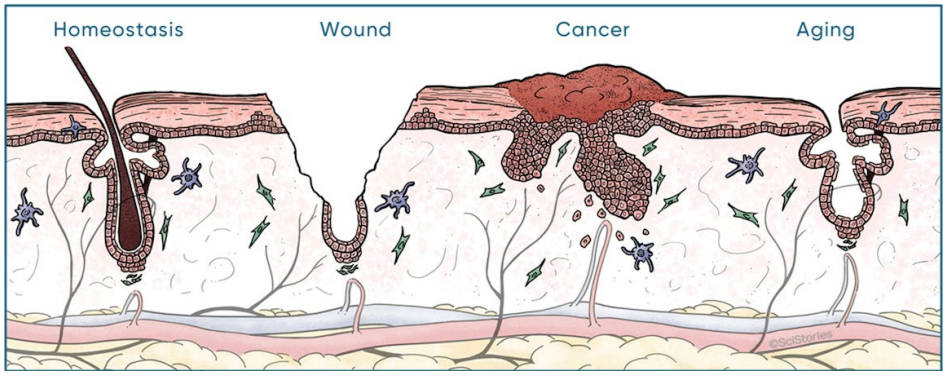
**Creativity**
Science is fun. Think outside the box.

**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.

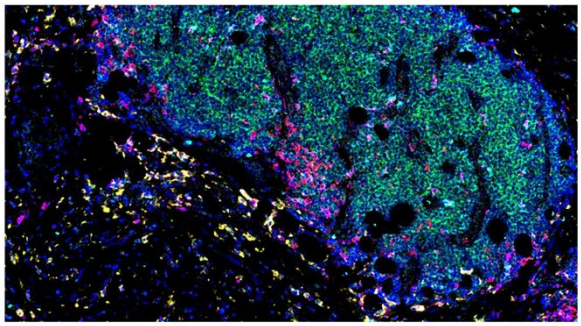
**Freedom**
You are here only because you want to be.

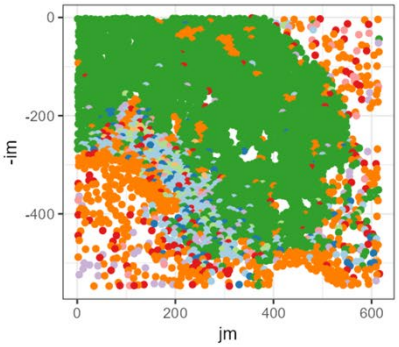


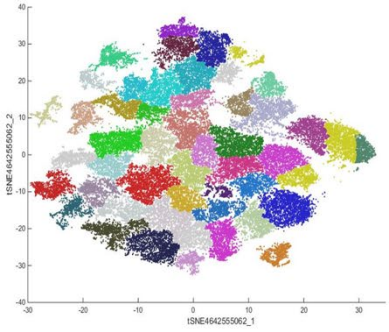

NATIONAL INSTITUTES OF HEALTH
THE UNIVERSITY OF TEXAS MD Anderson Cancer Center Making Cancer History
CPRIT
afar
WOMEN & SCIENCE
ROBERTSON FOUNDATION

# 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





**Ongoing and Future Research Projects**. Cancer cells don't grow in isolation; they constantly interact with the surrounding “normal” cells, including immune cells and fibroblasts. These interactions shape how tumors grow, spread, and respond to treatment. Understanding the “conversation” between cancer cells and the surroundings is key to creating better therapies. For example, some tumors keep immune cells (especially T cells, which can kill cancer cells) from entering the tumor area. If we learn how that happens, we might be able to develop new treatments that help immune cells get in and do their job. To this goal, we use cutting-edge tools such as single cell RNA-sequencing and spatial transcriptomics that let us profile each individual cell within a tumor mass, both in mice and in human samples, and the interactions between the cells. Then, we employ genetic tools and mouse models to functionally study the key players (*Tessaro et al., Cell Reports 2022*). Here are a few examples of the projects we’re working on in the Guarnerio Lab:

**Study how Cancer-Associated Fibroblasts (CAFs) affect tumor growth and immune exclusion.** We found that a certain type of fibroblasts, called glycolytic cancer-associated fibroblasts (or glyCAFs), gather around the edges of tumors and prevent T cells from getting into the tumor mass. They do this by sending out signals (like the Cxcr6/Cxcl16 pathway) that block immune cell movement. But when we disrupt this process, T cells can enter the tumor more easily, making treatments like chemo and immunotherapy to work better (*Broz et al., Nature Communications, 2024*). We're now working on understanding how these fibroblasts build up in tumors, what are their metabolic features, and how we can "reprogram" them to help, instead of harm, the immune response.

**Study tumor-promoting Circular RNAs**. Circular RNAs (circRNAs) are a special kind of transcripts that originate from back-splicing events. They’re very stable in the cells and can trick the immune system into ignoring tumors. Indeed, we’ve found that some of these circRNAs, like circCsnk1g3, repress interferon and inflammatory elements in the tumor cells, which are critical to make tumor cells visible to the cells of the immune system (*Piras and Ko, Nature Communications, 2022*). We’re now exploring how to block these circRNAs or design new ones that could boost the immune system’s ability to fight cancer. Moreover, we are expanding our knowledge on other tumor-related functions played by circRNAs. Our goal is to turn scientific discoveries into real treatments that attack cancer cells and their environment.

**The Guarnerio Lab**. Our lab was founded in 2018 in Los Angeles and moved to MD Anderson Cancer Center in 2024. We’re a diverse, collaborative group of scientists—PhD students, postdocs, and research assistants—who work together toward a common goal: improving cancer treatment through innovative research. *Learn more about us on our website!*

I’m passionate about training future scientists. I mentored several PhD students, helping them grow as independent thinkers and researchers. I believe graduate students play a vital role in science, and I'm committed to supporting them at every step of their journey here at MD Anderson. In our lab, PhD students lead their own projects with strong guidance and support from me and the team. We also help students apply for fellowships, develop grant-writing skills, and pursue the professional training they need to thrive.

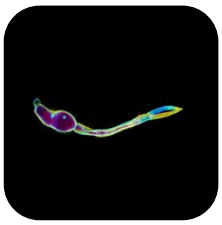
*If you're interested in working with our group, don’t hesitate to get in touch. We're happy to host one or two PhD students.*

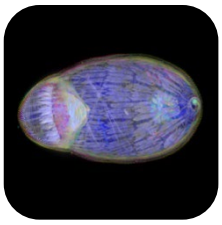
G&E Orientation 2025 – Jayhun Lee Lab

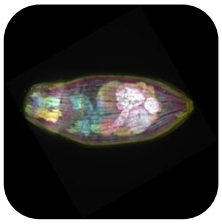
# Cell and developmental biology of parasitic flatworms in relation to survival and host immune evasion


## Jayhun Lee, Ph.D.

Assistant Professor
Department of Microbiology and Molecular Genetics
McGovern Medical School, UTHealth Houston
Faculty Member: GSBS MID (2021 – ); G&E (2025 – )
E-mail: Jayhun.Lee@uth.tmc.edu | Office: MSE R222
Webpage: sites.google.com/view/jleelabschisto
Social media: @jleelabschisto.bsky.social






**How does a multicellular organism thrive inside the bloodstream of a mammalian host for decades without being killed?** A parasitic flatworm *Schistosoma* (shis-tuh-SOH-muh) deploys robust stem cell-driven developmental programs to ensure proper intra-mammalian development, homeostasis, and reproduction. The primary focus of the Lee lab is to unveil the developmental mechanisms that are essential for the parasites to evade the host immune system and survive. Such discoveries may lead to novel strategies to target these parasites, which is urgently needed due to the disease they cause (i.e., schistosomiasis) that affects over 200 million people globally.

We use *S. mansoni* as a model and employ molecular and functional genomic tools, including (single-cell) transcriptomics, in situ hybridization, RNA interference, and high-resolution microscopy. We use mice as a mammalian host to introduce genetically perturbed parasites to dissect the mechanisms of gene function.

Our current work focuses on schistosomes' specialized secretory organ, the esophageal gland (EG), which is required for in vivo parasite survival and for blocking and degrading ingested host leukocytes (Lee et al., 2020). In particular, we are investigating the 1) regulation of cell-type heterogeneity governed by FoxA, a forkhead transcription factor essential for EG development and maintenance, 2) functions of FoxA co-regulatory factors in EG-associated tissues, 3) EG/FoxA-mediated systemic signaling of parasite stem cells, and 4) specific EG factors and their functions in immune evasion and survival.

'Team Schisto' is growing! We have two Ph.D students, Ryan Sloan (MID, post-candidacy) and Sabona Simbassa (MID, post-candidacy), and a Research Associate (Dr. Pallavi Yadav). Our trainees have received notable recognitions, including MBL Biology of Parasitism Course Fellowship, NIH R01 Diversity Supplement, Dr. John J. Kopchick Fellowship, and NRSA F31 Fellowship, just to name a few. The lab is supported by NIH (R01 AI175079, 2023 – 2028) and Dean's start-up funds. If you are fascinated by the biology of schistosomes or interested in studying the stem cell and developmental biology of a non-traditional model organism, feel free to connect with us.

UTHealth Houston | McGovern Medical School

**PI: Yonathan Lissanu MD, PhD**

Associate Professor
Department of Genomic Medicine
Department of Thoracic Surgery-Research
Contact: ylissanu@mdanderson.org
Website**: https://www.mdanderson.org/research/departments-labs-institutes/labs/lissanu-laboratory.html**

## Rewiring cancer cell epigenome using chemical induced proximity

Despite encouraging recent advances, most patients with advanced solid tumors lack effective therapeutic options, underscoring the dire need for additional treatment approaches. Genomic studies have identified frequent mutations in subunits of the SWI/SNF chromatin remodeling complex including *SMARCA4* and *ARID1A* in 20% of all solid tumors, making it the most frequently mutated complex in cancer. Unfortunately, there are no therapeutics approved for treating patients with these mutations. Research in the lab focuses identification of novel synthetic lethal genetic interactions to the SWI/SNF complex and discovery of novel chemical probes to rewire the epigenome as potential therapeutics. To this end we perform CRISPR-Cas9 functional genomic screens, utilize genetically engineered mouse (GEM) models and various epigenomic analysis (RNA-Seq, ATAC-Seq and CUT&RUN). These biological studies are supplemented by chemical biology approaches where we design and synthesize unique tool compounds to study chromatin biology and cancer cell epigenetics.

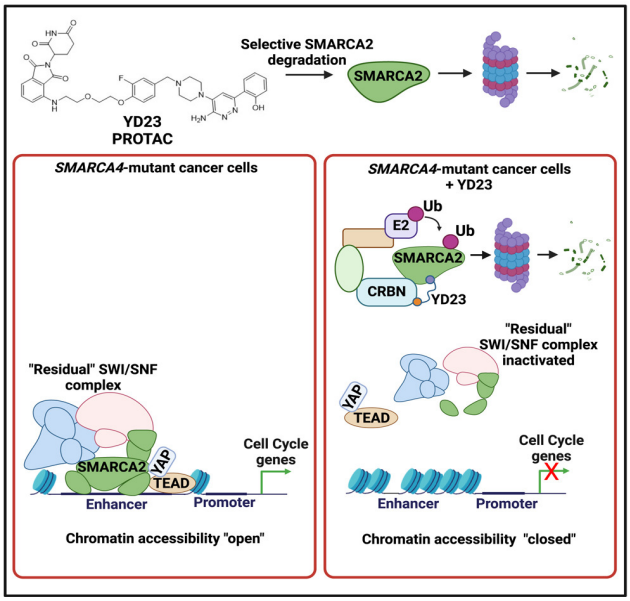



**Fig**. Targeted protein degradation can be used to rewire cancer cell epigenome for therapeutic benefit (from *Kotagiri…Lissanu, Cell Chemical Biology, 2024*).

For our chemical probe discovery efforts, we focus on an exciting and highly promising approach to modulate target proteins of interest using **induced proximity** concept. This approach enables us to **not only inhibit but eliminate cancer therapeutic targets as well as rewire cellular epigenome for therapeutic benefit**. So far, we have successfully designed, synthesize and characterized series of compounds targeting SMARCA2, a high value therapeutic target. We have published several papers on their discovery (Fig.) and patents covering these compounds are pending. These studies have the potential to provide **first-in-class cancer therapeutics for clinical development.**

**Environment**: We have a highly collaborative laboratory environment. Together with our collaborators and partners, we offer unique opportunities to learn drug discovery and development processes against epigenetic and chromatin targets including epigenomic profiling, target identification & validation, and drug target engagement assays. We look forward to inviting and advancing the scientific training of students interested in the intersection of **epigenetics, chemical biology and translational research**.

**People**: Nicholas (Nick) Blazanin (scientist), Sasi Kotagiri (postdoc), Mohamed Qudratullah (postdoc), Yanyan Han (postdoc), Xiaobing Liang (senior research associate), Vivian Dong (summer intern-undergraduate), Sami Shaikh (undergraduate)

# PI: Yuan-Hung Lo, Ph.D.

Assistant Professor
University of Texas MD Anderson Cancer Center
Department of Molecular and Cellular Oncology
Office: Z11.5042, Zayed Building
Contact: YLo1@mdanderson.org
Lab Website: https://www.mdanderson.org/research/departments-labs-institutes/labs/lo-laboratory.html

**Who We Are?**

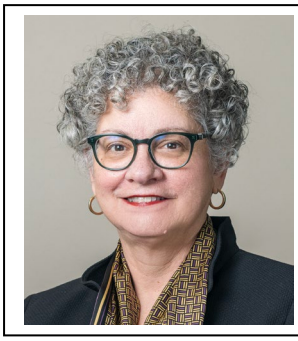
**The Lo Lab** is a dynamic and collaborative research team focused on understanding how genetic and epigenetic alterations regulate gastrointestinal (GI) stem cell function, cancer cell plasticity, and clonal evolution during tumorigenesis. We develop and apply advanced genetic tools and physiologically relevant 3D organoid models derived from primary human tissues and animal models to investigate the molecular and cellular mechanisms that drive cancer initiation, progression, and therapeutic response. Our ultimate goal is to develop innovative therapies to improve patient outcomes and quality of life. Our lab is currently focused on these areas:

- **Modeling Human Cancer in Primary 3D Organoids.** We are building innovative 3D tumor organoid models utilizing CRISPR/Cas9 genome-editing technologies. Our engineered tumor organoids faithfully mimic various stages of cancer progression. These models are valuable tools for investigating molecular mechanisms underlying tumor initiation, progression, and evolution.
- **Elucidating Cell States Dynamics in Cancers.** Human cancer development involves dynamic cell state changes and disruptions to signal pathways governing stem cell function and lineage differentiation. We are investigating tumor heterogeneity and therapeutic vulnerabilities of cancer cells using multi-omics single-cell technologies in our 3D organoid models. Our objective is to advance the understanding of cancer biology and develop new therapeutic strategies targeting cancer cell states.
- **Unveiling Gastrointestinal Stem Cell and Tumor Niche.** We are pioneering sophisticated 3D culture systems to simulate stromal components of the tissue microenvironment. Using state-of-the-art imaging technologies, we aim to unravel the intricate interactions between normal and malignant epithelial cells and their surrounding microenvironment. We seek to understand the multifaceted processes underlying normal physiology and malignancies.

**Welcome to joining the Lo lab!** Our team is dedicated to mentoring and supporting students. If you are passionate about research in gastrointestinal disease and want to be part of a team impacting the field, please get in touch with me!

**Selected Publications.** (Lo et al., *Nature Cancer* 2020) (Lo et al., *Cancer Discovery* 2021) (Lo et al., *Nature Communications* 2025)
Full list: https://www.ncbi.nlm.nih.gov/myncbi/yuan-hung.lo.1/bibliography/public/

PI/ Professor/Chair: **Guillermina Lozano PhD**
Dept Genetics, UT MD Anderson Cancer Center
Contact: gglozano@mdanderson.org



## The p53 tumor suppressor pathway

The *TP53* tumor suppressor encodes a DNA damage/stress response protein that functions as a transcription factor to activate numerous genes that prevent proliferation of damaged cells via initiation of cell cycle arrest and senescence, and via apoptosis and other mechanisms of cell death. Disruption of the pathway in tumors occurs most often through mutation or deletion of the *TP53* gene itself, and by elevated levels of two important p53 inhibitors, MDM2 and MDM4. At the *TP53* locus, missense mutations are the most common and these have additional activities that contribute to more aggressive and metastatic cancers. These have been studied in the germline mirroring the Li-Fraumeni syndrome and in three novel somatic mutant *Trp53* alleles that are wild type to start with but recombine the *Trp53* locus in a Cre-dependent manner to create a few mutant cells in a sea of normal stroma and immune cells. Somatic *Trp53* mutations in the breast epithelium cause breast carcinomas that metastasize to the lung and liver, common sites of human breast cancer metastasis. We are poised to decipher the changes that occur at each step of the metastatic process in this and other cancers by analyses of tumor evolution, circulating tumor cells, dormancy, and metastases.

We have explored the clinical implications of restoring p53 activity and deleting gain-of-function p53 mutants. Restoration of p53 has different effects in different contexts: tumor regression in tumors that lack *p53*; suppression of tumor growth but not tumor regression in mutant p53 tumors or in tumors with high Mdm2 levels. Additionally, deleting mutant *Trp53* in a breast somatic model leads to tumor regression through activation of ferroptosis.

Mdm2 deletion causes p53 dependent phenotypes in mice. We are using this system to physiologically reactivate p53 to functionally examine its transcriptional program and the downstream pathways that are activated on a single cell basis in various tissues. In addition, high MDM2 levels as observed in some human cancers are not tolerated by normal cells. We have an ongoing CRISPR/Cas9 screen to identify factors that allow normal cells to survive despite elevated levels of MDM2 to identify and characterize synthetic lethal relationships with high MDM2 in tumors.

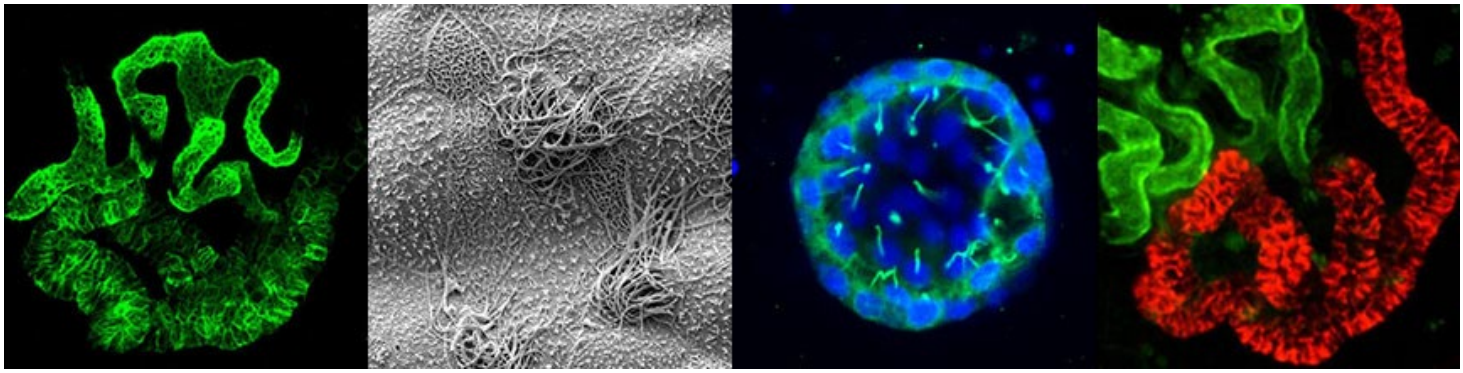
The ultimate goal of my laboratory is to understand the events that lead to tumor initiation, progression and metastasis, and to identify vulnerabilities that may be exploited in the clinic.

**Environment.** My laboratory consists of students, post docs, fellows and faculty. All work well together and discuss and share their data.

**Applying.** I would like to attract trainees who work independently with some direction.

# PI: Rachel K. Miller, Ph.D.

Associate Professor
Pediatric Research Center
McGovern Medical School, MSE R413
Contact: Rachel.K.Miller@uth.tmc.edu
Webpage: https://med.uth.edu/pediatrics/miller-lab/

## “Modeling kidney development and disease in frog embryos”

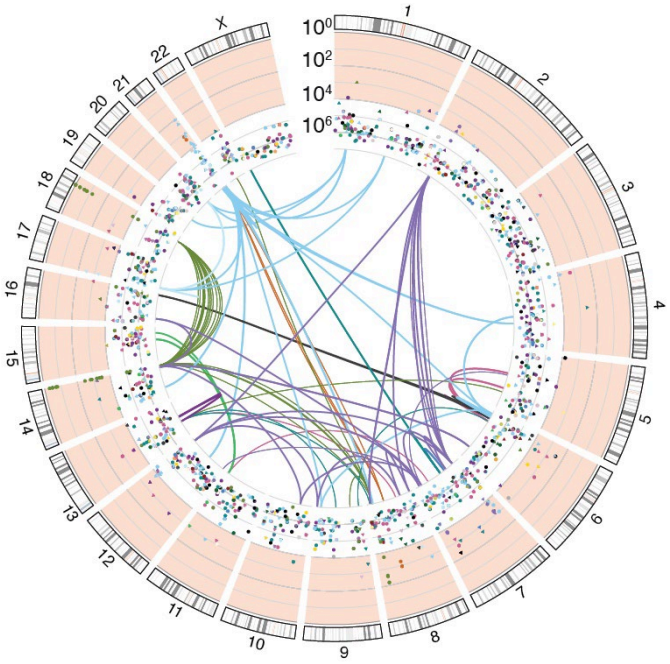
Our overall research goal is to understand the processes that underlie kidney development and how their disruption results in congenital anomalies of the kidney and urinary tract (CAKUT). Malformations of the kidney and urinary tract occur in almost 2% of the world population, representing nearly one-fourth 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, graduate students in our lab have been awarded 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, a Center for Clinical and Translational Sciences TL1 Fellowship, and the President’s Research Excellence Fund. 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.

**PI: Peter Van Loo, Ph.D.**
Professor and CPRIT Scholar in Cancer Research
Department of Genetics
Department of Genomic Medicine
The University of Texas MD Anderson Cancer Center
Basic Sciences Research Building, 15th floor
Contact: pvanloo@mdanderson.org

# Cancer Genomics and Evolution Laboratory




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 and succesfully complete their PhDs in 4-5 years.

**Applying**

Our lab is growing, funded by a $6 million CPRIT award. 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

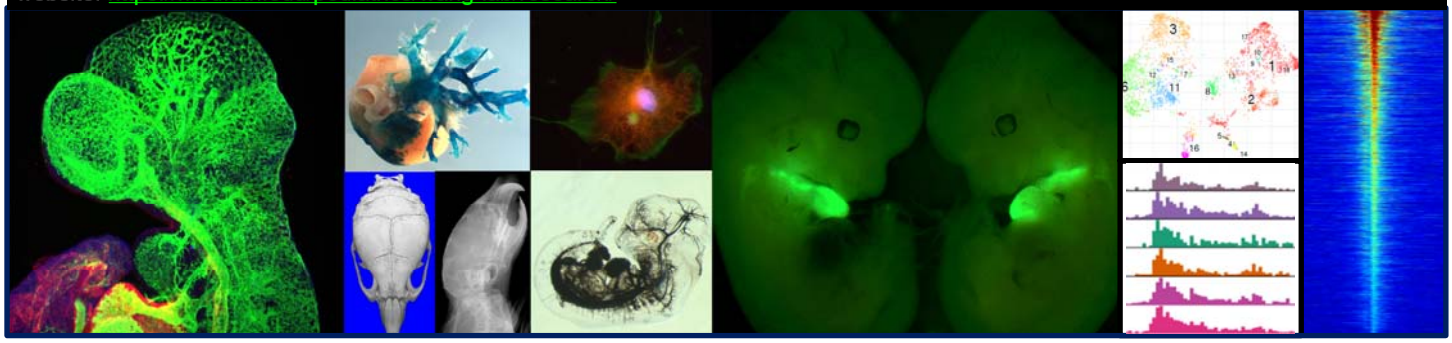
# PI: Jun Wang, PhD

Associate 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 Research Interests:**

**"Molecular regulation of heart and head development, diseases and regeneration"**

website: https://med.uth.edu/pediatrics/wang-lab/research/



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.

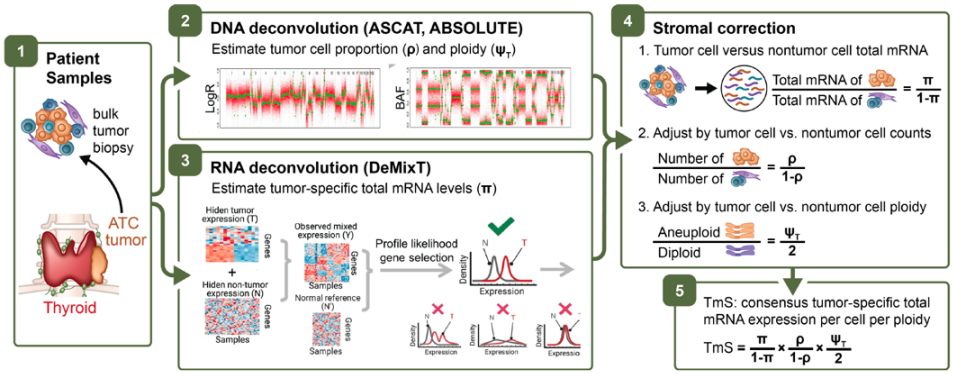
**Wang Lab Environment**. Wang lab is a highly collaborative team, consisting of postdoctoral fellows, graduate students, research associates, research assistants, and undergraduate researchers. Our group is actively seeking students. The PI has been devoted to mentoring trainees and helping them to reach their career goals. Trainees actively attend national or international meetings and have received numerous honors and awards. 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 students about the Wang lab: **Shannon Erhart** (NIH F31 Award recipient) and **Julianna Quinn** (AHA predoctoral fellowship recipient).

**PI: Wenyi Wang, Ph.D.**
Professor
Department of Bioinformatics and Computational Biology
The University of Texas MD Anderson Cancer Center
7007 Bertner Ave. Floor 12, 1MC12.2240
Contact: WWang7@mdanderson.org

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 across diverse cancer types. 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 testable hypotheses and 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 been making pioneering contributions to developing methods and software tools for the studies of tumor heterogeneity and tumor evolution, such as MuSE for subclonal mutation calling (***Fan Y, et al., Genome Biol 2016***), with the recent MuSE 2.0 offering a 50-fold increase in speed (***Ji S, et al., Genome Res. 2024***); and a pan-cancer characterization of genetic intra-tumor heterogeneity in subclonal selection (***Dentro S, et al., Cell 2021***). Concurrently on the forefront of studying cancer transcriptomics, we developed DeMixT for cancer-cell specific transcriptome 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 for cancer prognosis and treatment response (***Cao S, et al., Nat Biotechnol 2022,*** *see Illustration above*). Most recently, we introduced DeMixSC, a framework that leverages single-cell RNA sequencing and a small benchmark dataset to accurately estimate cell type proportions from bulk RNA-seq in complex tissues (***Guo S, et al., Genome Res. 2025***).

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 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, Nguyen NH, et al., JCO CCI 2024***). 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 been published in prestigious medical/biological journals and top statistics journals, e.g., *JCO*, *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.

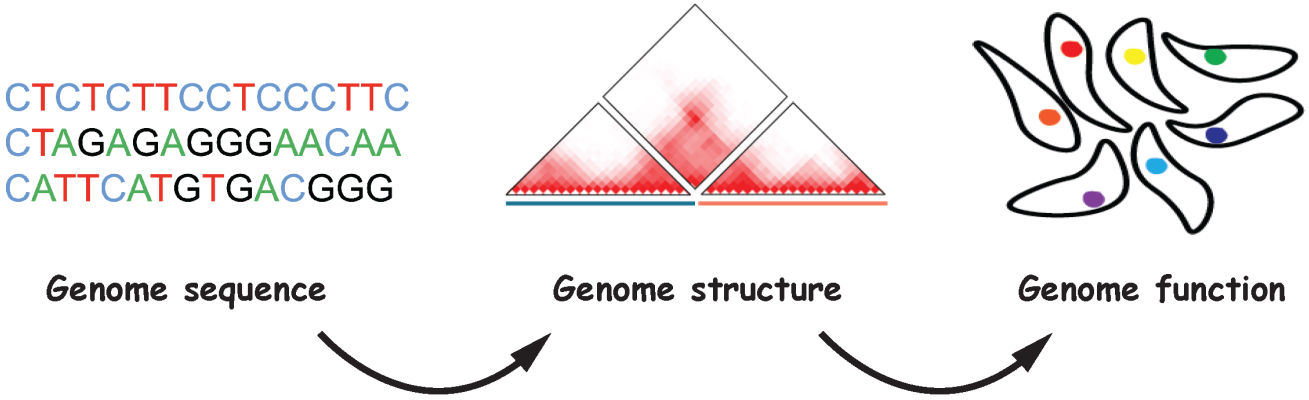
## Applying

We are recruiting similar-minded and enthusiastic PhD or master students who are interested in innovative analysis and interpretation of clinical data and/or multi-omic data using computational tools, as well as therapeutic biomarkers and new treatment strategy development.

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

# PI: Zhichao Xu, Ph.D.

Assistant Professor
Department of Biochemistry and Molecular Biology
McGovern Medical School, MSB 6.126
Contact: zhichao.xu.1@uth.tmc.edu
Webpage: https://zxulab.org/




## Modeling Function of Structural variants in Human Cancer

**Can we predict gene dysregulation when a nearby structural variant is observed?**

In this project, we will explore the functional consequences of rearrangements in the human genome. We will first engineer complex rearrangements near the oncogene of interest. We will then simultaneously measure gene expression and the variant event by utilizing single-cell multi-omic sequencing, followed by computational modeling. This will first identify risky partner regions genome-wide for certain oncogenes, then generate an oncogene expression prediction model for cancer diagnosis in the future.

**What is the mechanism of distal gene regulation, and how is it reorganized in cancer and evolution?**

In this project, we will investigate the rules of distal enhancer-promoter communications. We will first develop a genome conformation perturbation assay to identify specific cis-motifs required for distal enhancer-promoter looping and the regulation of the gene of interest. Further exploration of the prevalence of such elements in super-enhancers and oncogene promoters, as well as their corresponding trans-factors, will provide insights into the enhancer hijacking mechanism in cancer and genome evolution across different species.

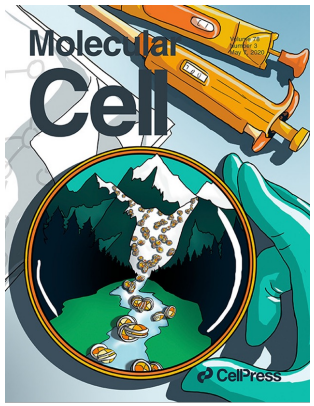
**Applying.** Our lab is growing! Please get in touch with me if you are interested in working with our group. We wish to recruit one new graduate student who is interested in getting trained in both genetics and bioinformatics!

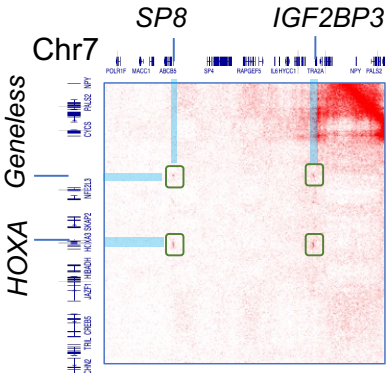
# Epigenetic regulation and targeting in acute myeloid leukemia
## Xiaotian Zhang lab

UTHealth Houston
McGovern Medical School

3D genome organization of *stem cell and primary human cancer*.

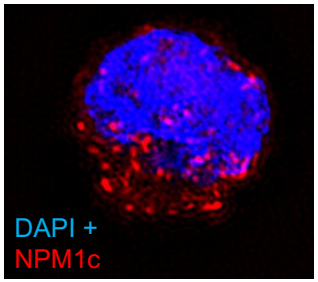
New epigenomic biomarker for precision medicine of EZH2 inhibitor in pan-Cancer: Long Polycomb loop .

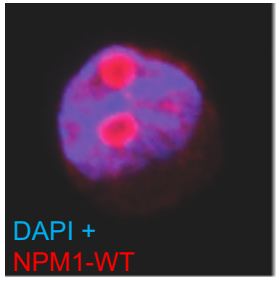


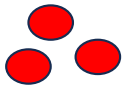

Zhang et al, 2020

Develop small molecules inhibitor targeting the transcriptional hijacking Condensate in cancer




Wang et al, Cancer Discovery, 2023



Condensate
Of oncogenic
Mutant protein

- Alteration of condensate Property
- Disrupt the condensate formation


Lab: UTHealth MSB6.006
Email: xiaotian.zhang@uth.tmc.edu

***Seeking 2-3 Graduate Students***

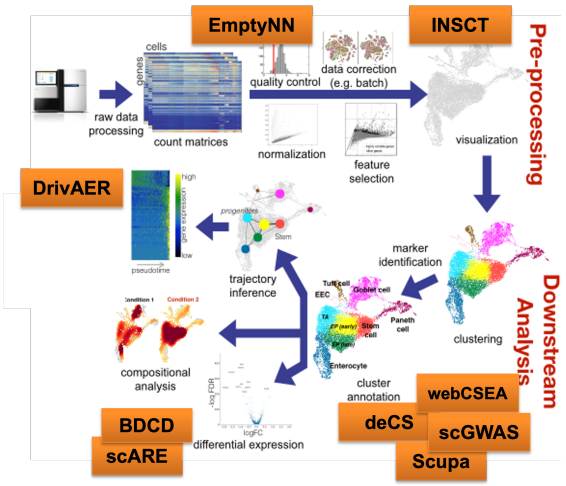
# PI/ Professor: Zhongming Zhao, PhD

Vice President of Cancer Genomic Medicine, Chair Professor for Precision Health, McWilliams School of Biomedical Informatics, UTHealth
Contact: zhongming.zhao@uth.tmc.edu

## "Multi-Scale, Integrated, and Contextualized (MUSIC) Approaches for Complex Disease Study"

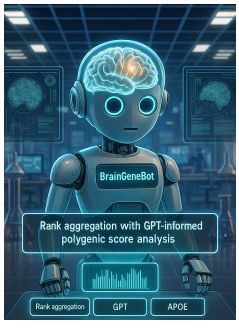
The lab focuses on genomics, deep learning, precision medicine, translational science, and big data science. We develop novel computational approaches for deep understanding genetic variants and regulatory mechanisms in complex disease. Deep learning and single-cell omics approaches are often applied. Representative projects are below. The students may work with Dr. Zhao to identify a long-term project for thesis.

**Multi-Scale, Integrated, and Contextualized Approaches for Complex Disease.** In this NIH U01 project, we will develop and implement a robust AI framework, namely AIM-AI, for transforming the genetic catalog of Alzheimer's disease (AD) in a way that is Actionable, Integrated and Multiscale. We will engineer novel deep learning algorithms and GPT-based foundational models to build a powerful brain molecular chronological age predictor and further to dissect cell-type specific, genetic regulatory mechanisms in complex disease like AD.




Single-cell methods/tools in Zhao lab.

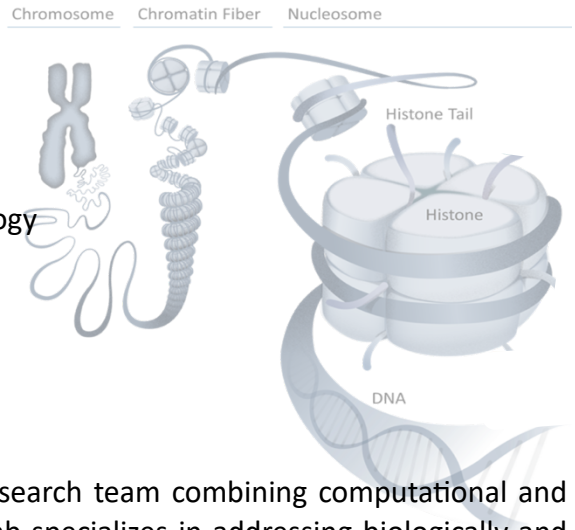
**Predicting Phenotype by Deep Learning Heterogeneous Multi-Omics Data.** In this renewed NIH R01 project (since 2012), we combine multi-omics, phenotype, AI models, and electronic medical record (EMR) data mining to develop novel analytical strategies that maximally leverage regulatory information in phenotype prediction.




**Single-cell Omics Approaches for Neurodevelopmental Diseases and Cancer Immunotherapy.**

**Environment**. The Bioinformatics and Systems Medicine Laboratory (BSML, web: uth.edu/bioinfo), directed by Dr. Zhao, has been very productive since it was originally founded in Vanderbilt University Medical Center in 2009 and later moved to UTHealth in 2016, with more than 400 publications in the past 15 years. The lab is in an interdisciplinary research environment, currently with 19 members. All the PhD students in Zhao lab since 2016 have received fellowships or scholarships. The lab has developed many computational methods/tools and biomedical databases/resources.

**Applying**. We encourage those students who are interested in deep learning, genomic medicine and single-cell omics to apply. We have stable and abundant funding including several NIH/CPRIT grants as well as Dr. Zhao's startup and chair professorship fund.




**Ye Zheng, Ph.D.**
Assistant Professor
Department of Bioinformatics & Computational Biology
Department of Systems Biology
M.D. Anderson Cancer Center
Email: yzheng8@mdanderson.org
Compbio Wizard Group Website:
https://compbiowizard.github.io./

The Compbio Wizard Group is a dynamic hybrid research team combining computational and experimental approaches. Our computational dry lab specializes in addressing biologically and clinically significant challenges through innovative statistical models, traditional and modern computational methods, and integrative multi-omics data analysis. Our research spans diverse topics leveraging rich multi-modality data, including **epigenomics** (Henikoff and Zheng et al, Science, 2025), **3D genomics** (Zheng and Keles, Nature Methods, 2020), **proteomics** (Zheng and Caron et al, Nature Communications, 2025), and their applications in **cancer** studies (Zheng et al, PNAS, 2025), CAR-T cell **immunotherapies** (Fiorenza and Zheng et al, Nature Communications, 2024), Alzheimer's disease and other disease systems. The wet lab focuses on **epigenomic profiling of FFPE** samples, unraveling gene regulation mechanisms across human cancers, as highlighted in our latest *Science* study.

The group members work both independently and collaboratively, leading their own topics while sharing skills and benefiting from others' results. The group fosters an energetic atmosphere that encourages enjoyment of science while motivating innovation for scientific discoveries.

**Keywords: Bulk and Single-cell Multi-omics Study in Cancer**

- Epigenomics
- Proteomics
- 3D genomics
- Transcriptomics
- Cancer and Immunotherapy
- Statistics, Computational Algorithm, Machine Learning, AI Pathology