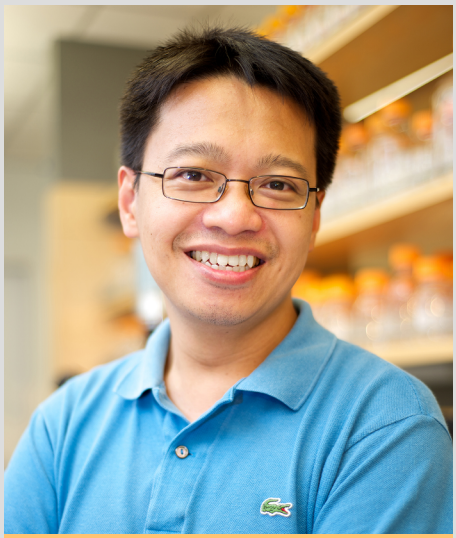


VANDERBILT SCHOOL OF MEDICINE | Basic Sciences

# Manuel Ascano, PhD


**Assistant Professor of Biochemistry**
**Assistant Professor of Pathology, Microbiology and Immunology**

***manuel.ascano@vanderbilt.edu***
***615-875-8714***
***https://lab.vanderbilt.edu/ascano-lab***


## Biosketch

**Manny was raised in New York City and Chicago and received his Bachelor's in Microbiology from the University of Illinois Urbana-Champaign. He received his Ph.D. in Biochemistry from the University of Cincinnati, focusing on a pathway important to cancers. Manny trained as a postdoctoral fellow at Rockefeller University in New York City where he gained expertise in RNA biochemistry and innate immunity. He joined Vanderbilt in 2014 as a faculty member of the Department of Biochemistry. Among his research contributions, he is also an inventor of a patent for a molecule that can activate the immune system, which has relevance to the development of immunotherapeutics for autoimmune disorders and cancers.**

## Key Publications


**"Small molecule inhibition of cGAS reduces interferon expression in primary macrophages from autoimmune mice," *Nature Communications*, Sep 29;8(1):750, 2017**

**"Cyclic [G(2',5')pA(3',5')p] is the Metazoan Second Messenger Produced by DNA-Activated Cyclic GMP-AMP Synthase, *Cell*, 153(5):1094-1107", 2013**

**"FMR1 targets distinct mRNA sequence elements to regulate protein expression," *Nature*, 492(7249):382-386, 2012**

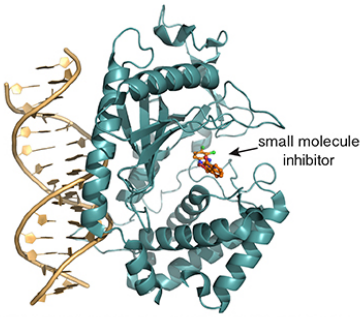

## "Understanding innate immunity to inspire new therapies for infectious disease, autoimmunity, and human cancers"

The Ascano lab integrates Biochemical and Chemical Biological approaches with High-throughput Transcriptomic and Proteomic technologies to **investigate the impact of RNA and DNA binding proteins in innate immunity, viral infection, and human disease**. Current research activities include:

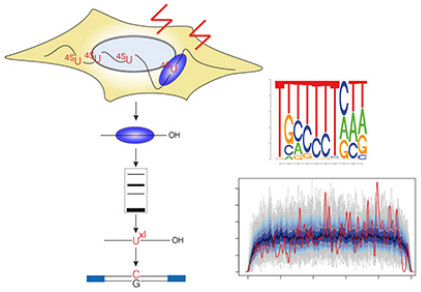
cGAS is a critical protein required for sensing foreign or damaged cellular DNA. Can we **understand the mechanism of cGAS enzyme activity** by designing small molecule activators and inhibitors, and can these compounds develop into novel drugs **for treatment of autoimmune disorders or cancers?**

RNA binding proteins play essential roles in regulating gene expression; without them, no protein can be made. **How do RNA binding proteins like ELAVL1 and IFIT1 regulate cellular and viral RNAs to promote cell survival during infection?**

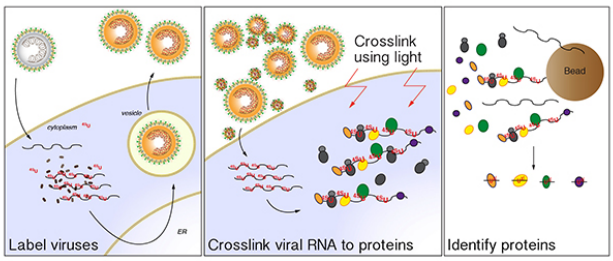
What human proteins make the first contacts with viral RNA during an infection? And **what role do these proteins play?** Anti- or pro-viral? Over 160 viruses that infect humans contain an RNA genome, and they include: **Influenza, HIV, SARS, and Ebola.**




The protein cGAS recognizes foreign DNA and triggers an inflammatory response. Small molecules can target its activity.



PAR-CLIP technology identifies all gene targets of RNA binding proteins like ELAVL1 and IFIT1 to determine which are important for immunity and cell survival.




VIR-CLASP technology captures the earliest events between viral genomes and human cells during infection. 73% of all viruses that cause human disease contain RNA genomes.

VANDERBILT SCHOOL OF MEDICINE | Basic Sciences

# Breann Brown, PhD

**Assistant Professor of Biochemistry**


***breann.brown@vanderbilt.edu***
***615-343-1632***
***https://lab.vanderbilt.edu/brown-lab***


## Biosketch

**Dr. Brown was born and raised in the DC metro area. To pursue her love for research, she received her Ph.D. from Brown University in Providence, RI where she investigated protein pairs that play a role in bacterial multidrug tolerance and chronic biofilm infections. She then completed her postdoctoral training at the Massachusetts Institute of Technology where she used X-ray crystallography and biochemical techniques to study mechanisms of protein assembly in both bacteria and human metabolic systems. Arriving at Vanderbilt in 2019, her lab uses structural biology to understand how mitochondrial proteins assemble to maintain human health.**

## Key Publications


**"Structure of the Mitochondrial Aminolevulinic Acid Synthase, a Key Heme Biosynthetic Enzyme," (2018) *Structure*, 26, 580-589**

**"N domain of the Lon AAA+ protease controls assembly and substrate choice," (2018) *Protein Science*, doi: 10.1002/pro.3553**

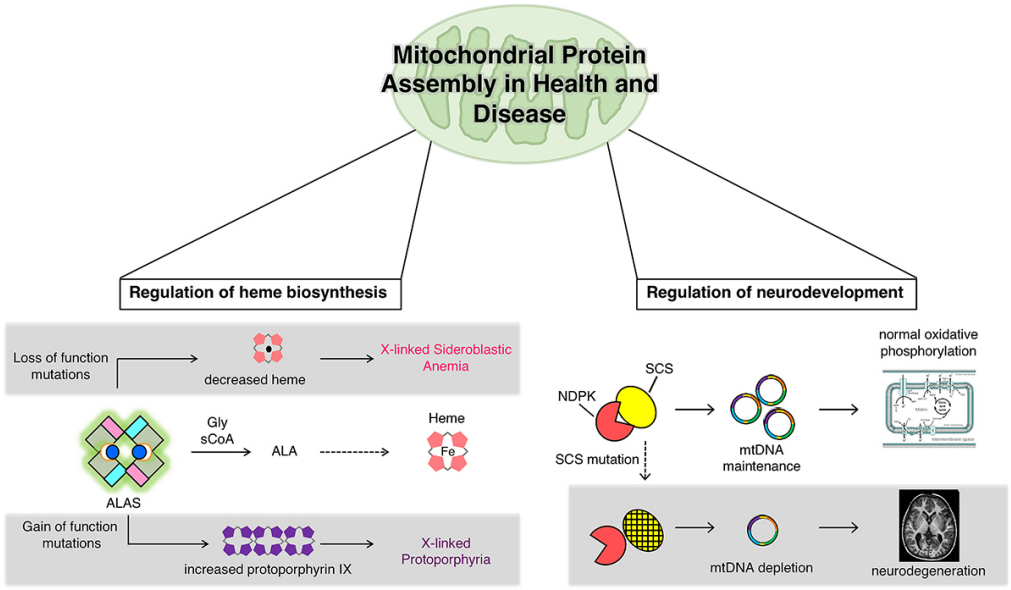
**"Structure of the E. coli antitoxin MqsA (YgiT/B3021) bound to its gene promoter reveals extensive domain rearrangements and the specificity of transcriptional regulation," (2011) *J. Biol. Chem.* 286, 2285-2296**


## "Understanding mitochondrial protein assembly in human health and disease"

The focus of the Brown Lab is to investigate **3-dimensional protein structure** in order to understand how certain **genetic mutations** can have profound impact on human health.

In several instances, proper **protein complex assembly** is critical for maintaining human health by modulating various **cellular processes** such as activity of signaling pathways, providing feedback regulation, and mediating transport and transfer of molecules among partners. Unfortunately, there are numerous painful, debilitating, and life-threatening diseases that occur due to genetic mutations that prevent proper protein assembly. Our approach is to use **X-ray crystallography** and other complementary biochemical techniques **to understand how these various mutations lead to changes in protein structure,** which is tightly correlated to protein function, thus preventing **proper macromolecular assembly**.

**We focus on areas of human health related to mitochondrial biology and metabolism**. Specifically, we seek to understand **assembly mechanisms responsible for regulation of heme biosynthesis**, which is altered in several blood diseases, and **maintenance of mitochondrial DNA copy number,** which has direct implication in proper neuronal development. In the future, our work will lay the foundation for **developing therapeutics** that may take advantage of previously unknown cellular avenues.



VANDERBILT SCHOOL OF MEDICINE | Basic Sciences

# Dylan Burnette, PhD

**Assistant Professor of Cell and Developmental Biology**
**Vanderbilt University School of Medicine**

***dylan.burnette@vanderbilt.edu***
***410-925-7705***
***https://lab.vanderbilt.edu/dylan-burnette-lab***

## Biosketch

**Dr. Dylan Burnette was born and raised in Dalton, GA. He attended Georgia public schools through his bachelor's degree from the University of Georgia, Athens. At UGA, Dylan became enamored with the question of how cells change their shape in order to move.**

**Shape-change is inherently a property of a cell's cytoskeleton. As such, the cytoskeleton has been the focus of Dylan's research interests his entire adult life. He has previously perused these interests during his graduate work at Yale University studying neurons and his post-doc at the National Institutes of Health studying cancer cells.**

## Key Publications

**"Focal adhesions control cleavage furrow shape and spindle tilt during mitosis," *Scientific Reports*, Jul 19;6:29846, 2016**

**"Expansion and concatenation of non-muscle myosin IIA filaments drive cellular contractile system formation during interphase and mitosis," *Molecular Biology of the Cell*, mbc.E15-10-0725; First Published on March 9, 2016**

**"A contractile and counterbalancing adhesion system controls the 3D shape of crawling cells," *Journal of Cell Biology*, Apr 14;205(1):83-96, 2014**

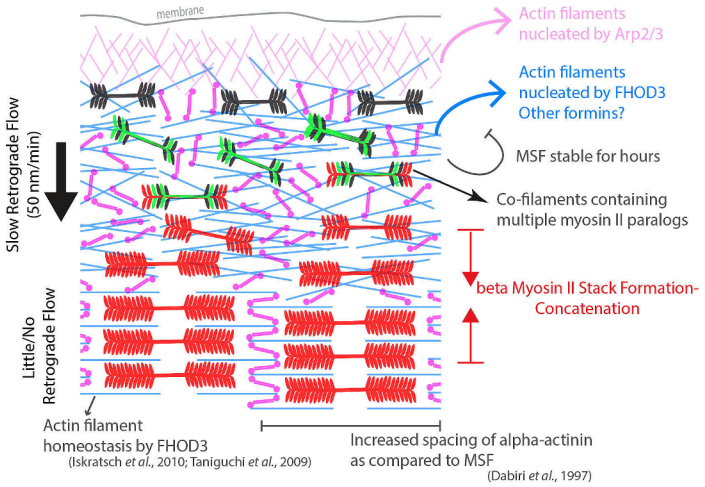
## "Assembling contractile systems to drive cell motility and muscle contraction"

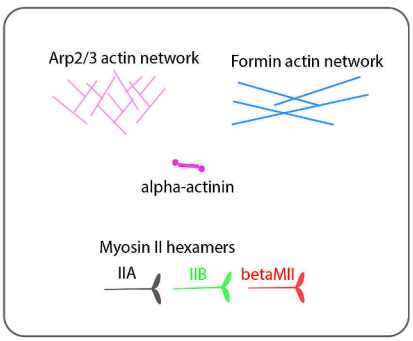
Our cells produce **contractile force**. This type of cellular force is usually a good thing. Currently, our **heart muscle cells** are producing contractile force to pump blood throughout our bodies. Our **immune cells** can use contractile forces to hunt down and kill external threats (e.g., bacteria) and internal threats (e.g., cancer cells). However, when gone awry, **contractile forces can result in catastrophic health changes**.

Abnormalities in contractile forces lead to **heart disease**, and **cancer cells also utilize contractile forces to move away from a primary tumor** (i.e., metastasize). Research in the Burnette lab revolves around the **molecular motor that generates contractile force: myosin II**. Different versions of myosin II drive cell crawling, cell division, and muscle contraction. As such, **we study the function of myosin II** in these three cellular contexts.

Currently, we are fascinated with **how myosin II-based contractile systems assemble within cells**. By combining high resolution microscopy, high content microscopy, and genomic/proteomic analysis, we are working out the details of assembly. Our long-term goal is to determine how we can **re-assemble diseased contractile systems**.

### Sarcomere Assembly in Human Cardiomyocytes



VANDERBILT SCHOOL OF MEDICINE | Basic Sciences

# Erin Calipari, PhD


**Assistant Professor of Pharmacology**
**Assistant Professor of Psychiatry and Behavioral Sciences**
**Assistant Professor of Molecular Physiology & Biophysics**

***erin.calipari@vanderbilt.edu***
***615-343-5792***
***http://caliparilab.com/erin-s-calipari***


## Biosketch

**Dr. Calipari received her PhD in Neuroscience in from Wake Forest University School of Medicine where she studied how self-administered drugs altered dopaminergic function to drive addictive behaviors. She then completed her postdoctoral training at Icahn School of Medicine at Mount Sinai, where she used circuit probing techniques to understand the temporally specific neural signals that underlie motivation and reward learning in behaving animals. Work in her lab integrates microscopy with molecular tools to record neural activity in the brain during behavior and determine the molecular targets that underlie changes in neural activity in response to environmental stimuli.**

## Key Publications


**"Granulocyte colony stimulating factor enhances reward learning through potentiation of mesolimbic dopamine system function," *J. Neuroscience*, In Press, 2018**

**"Dopaminergic dynamics underlying sex-specific cocaine reward," *Nature Communications*, 8:13877, 2017**

**"In vivo imaging identifies temporal signature of D1 and D2 medium spiny neurons in cocaine reward," *Proceedings of the National Academy of Sciences U.S.A.*,113(10):2726-31, 2016**


## How does the brain encode information on a cellular level, and how does dysregulation in this process underlie psychiatric disease?

**Our research is guided by two overarching questions:**

1. How do **neural circuits integrate experiences** with positive and negative stimuli to guide behavior?

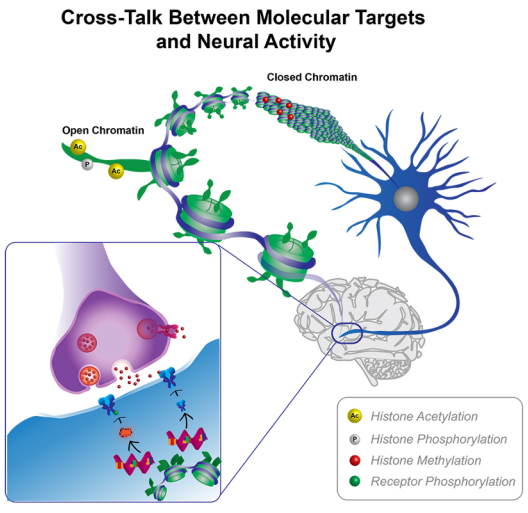
2. How does **dysfunction** in this process **precipitate psychiatric disease**?

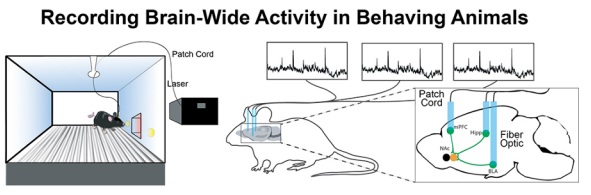
One of the most fundamental forms of learning is the ability to associate **positive and negative stimuli** with cues that predict their occurrence. The ability to seek out rewarding, and avoid negative, stimuli is critical to survival and is evolutionarily conserved across species. However, **dysregulation** of these processes can precipitate a number of psychiatric disease states.

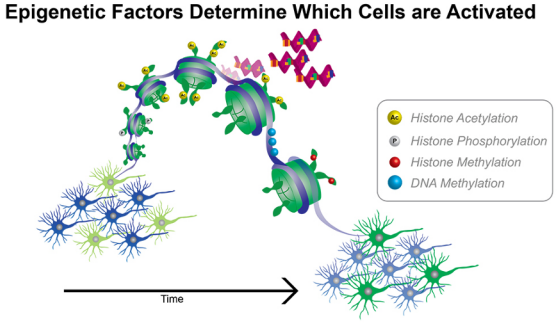
**Addiction, depression, and anxiety** are all examples of syndromes characterized in part by dysregulation of associative learning. These are among the most prevalent neuropsychiatric disorders and are highly comorbid. Therefore, **understanding the neural mechanisms governing associative learning** has widespread implications for developing treatment interventions for psychiatric disease.

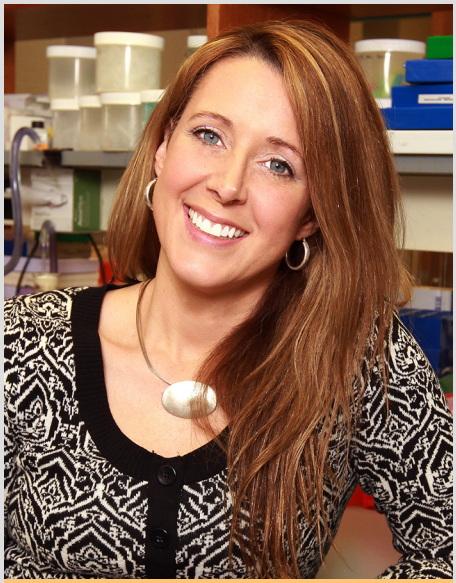
Our work aims to combine cutting-edge technology with comprehensive models of psychiatric disease to **understand the circuit and molecular dysregulation** that underlies these disorders.

Together, our work uses and develops cutting-edge techniques to **outline the precise cells in the brain that encode information** and push the boundaries of how we understand learning, memory, and disease.





VANDERBILT SCHOOL OF MEDICINE | Basic Sciences

# Rebecca Cook, PhD


**Associate Professor of Cell & Developmental Biology**
**Associate Professor of Biomedical Engineering**
**Breast Cancer Research Program, VICC**

***rebecca.cook@vanderbilt.edu***
***615-636-3148***
***https://my.vanderbilt.edu/rebeccacooklab/***


## "How tumors manipulate the immune system to increase cancer spreading throughout the body"

The research goals of the Cook laboratory are to **understand molecular pathways regulating breast development**, how these are **commandeered by cancers**, and to apply this information towards **developing stronger, safer treatments** for breast cancer patients.

Recent efforts are aimed at understanding how **immune responses in the tumor microenvironment become suppressed**, creating a permissive and fertile soil for tumor progression. Importantly, we are finding ways to a**lter immune responses in breast tumors** to fight tumor cells at all stages of cancer progression.

### Biosketch

**Dr. Cook was born and raised in rural Alaska, received her Bachelor's degree in Biology from Vanderbilt University in 1993 and a Ph.D. in Developmental Biology from the University of Cincinnati in 1998. Dr. Cook did her post-doctoral fellowship training in the field of breast cancer biology with Dr. Carlos Arteaga at Vanderbilt University and with Dr. H. Shelton Earp at University of North Carolina. Dr. Cook joined the faculty at Vanderbilt University in 2010.**

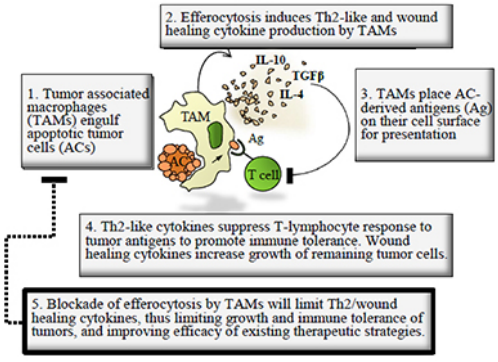
### Key Publications


**1. "ErbB3 downregulation enhances luminal breast tumor response to antiestrogens," *Journal of Clinical Investigation*, Volume 123, pages 4329-4343. 2013**

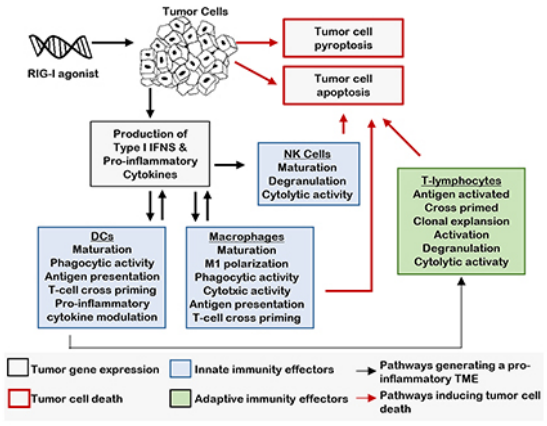
**2. "Efferocytosis produces a prometastatic landscape during postpartum mammary gland involution," *Journal of Clinical Investigation*, Volume 124: pages 4737-4752. 2014**

**3. "Treatment-induced tumor cell apoptosis and secondary necrosis drive tumor progression in the residual tumor microenvironment through MerTK and IDO-1," *Cancer Res*, epub ahead of print. PMID: 30413412, 2018**


How does tumor cell death suppress immune responses in tumors?




How can we re-direct communication between the dying tumor cell and the immune system?



VANDERBILT SCHOOL OF MEDICINE | Basic Sciences

# James Dewar, PhD

**Assistant Professor of Biochemistry**
**Vanderbilt University School of Medicine**

***james.dewar@vanderbilt.edu***
***615-875-8125***
***https://lab.vanderbilt.edu/dewar-lab***

## Biosketch

**Dr. Dewar carried out his undergraduate studies at the University of Bath, England, and graduated in 2007 with First Class Honours in Molecular and Cellular Biology. He received his PhD in 2011 in yeast genetics from Newcastle University, England, before undertaking post-doctoral training in Johannes Walter's lab at Harvard Medical School, where he became a Charles A. King Trust Fellow. In 2016, Dr. Dewar joined the Vanderbilt Faculty as an Assistant Professor of Biochemistry.**

## Key Publications

**"Mechanisms of DNA replication termination,"** ***Nat Rev Mol Cell Biol*****, 18, 507-516, 2017**

**"CRL2Lrr1 promotes unloading of a vertebrate replisome from chromatin during replication termination,"** ***Genes Dev*****, 31, 275-290, 2017**

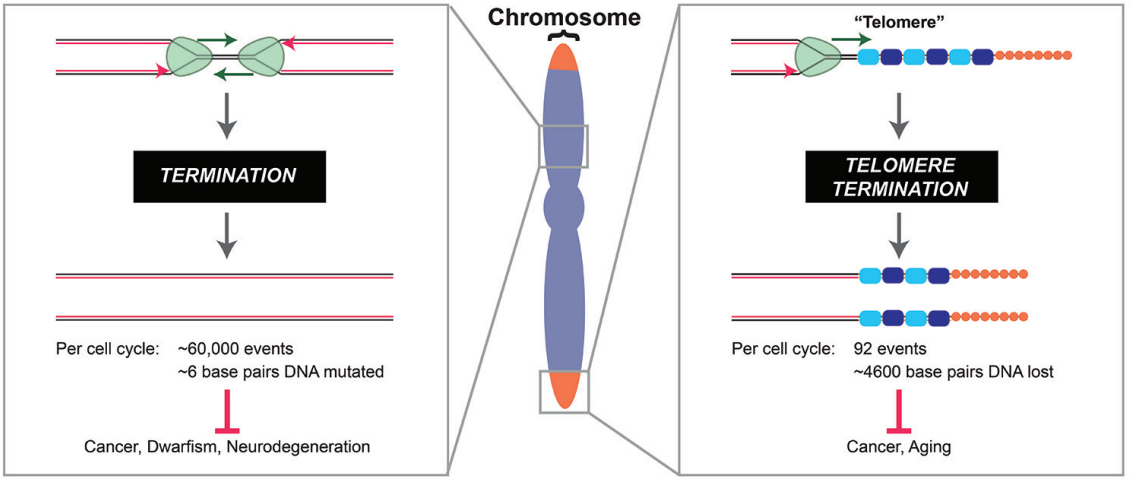
**"The Mechanism of replication termination in vertebrates,"** ***Nature*****, 525, 345-50, 2015**

## "DNA Endgame: Using frog egg extracts to unravel how copying of the DNA blueprint is terminated"

**DNA inside a human cell is faithfully replicated** with an error rate of ~1 in a billion. The fidelity of this process is critical **to prevent a diverse set of diseases, from cancer, to dwarfism and neurodegeneration**. DNA replication in humans involves loading and activation of ~60,000 DNA replication machines which copy the DNA.

**Completion of DNA replication is called termination** and occurs when pairs of copy machines meet head-on upon the same stretch of DNA. **Termination is highly perilous in bacteria and viruses** and was assumed to be equally-problematic in humans. This assumption persisted for decades, because technical limitations prevented termination from being studied in cells. However, using a 'cell in a tube' approach derived from frog egg extracts, **Dr. Dewar showed that termination in humans is rapid**, suggesting humans possess specific proteins to promote termination.

**The Dewar lab is working to identify proteins that promote rapid termination**. It is particularly important to study termination **because this process is targeted during chemotherapy**. The Dewar lab is also working to understand a specialized form of termination that occurs at **telomeres**, which cap chromosome ends and **impair cellular aging**.



VANDERBILT SCHOOL OF MEDICINE | Basic Sciences

# Vivian Gama, PhD

**Assistant Professor of Cell and Developmental Biology**
**Vanderbilt School of Medicine**

***vivian.gama@vanderbilt.edu***
***615-875-9490***
***https://lab.vanderbilt.edu/gama-lab***

## Biosketch

**Dr. Gama was born in Bogotá, Colombia where she did her undergraduate work at the Universidad de Los Andes. Vivian received her Ph.D. in Pharmacology from Case Western Reserve University. She did her postdoctoral training with Dr. Mohanish Deshmukh at the University of North Carolina at Chapel Hill. She joined the faculty at Vanderbilt University in 2015. Her laboratory has received funding from NCI/NIH, NIGMS/NIH, ABTA and the AHA.**

## Key Publications

**"A non-apoptotic function of MCL-1 in promoting pluripotency and modulating mitochondrial dynamics in stem cells,"** ***Stem Cell Reports*****, 10, Issue 3, p684–692, 2018**

**"PARC/Cul9 Mediates the Degradation of Mitochondrial-released Cytochrome c,"** ***Science Signaling*****, 7, ra67, 2014**

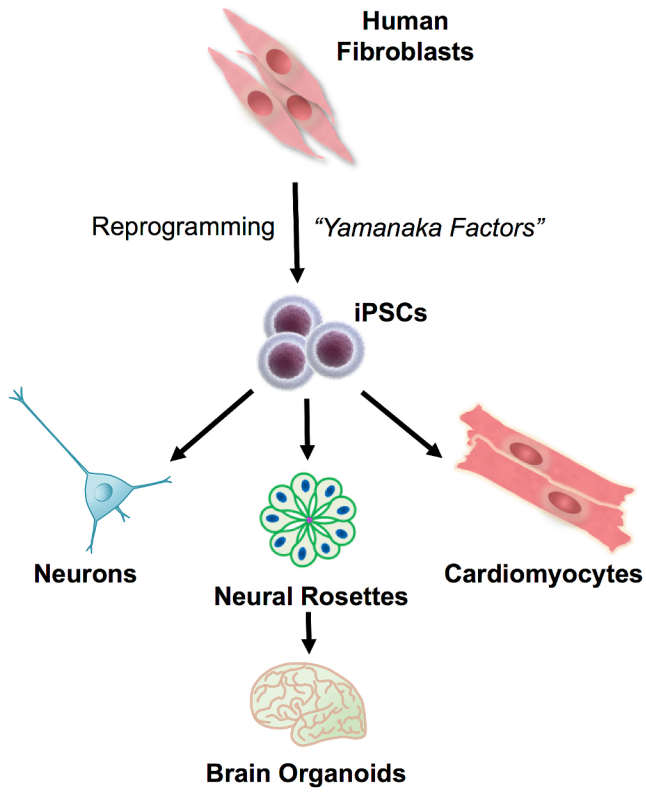
**"Human Embryonic Stem Cells have Constitutively Active Bax at the Golgi and are Primed to Undergo Rapid Apoptosis,"** ***Mol. Cell.*****, 46: 573-583, 2012**

## "Tipping the balance in the powerhouse of the cell to kill brain tumors "

We conduct our research using an **innovative interdisciplinary approach** that combines cellular, biological, and biochemical assays with state-of-the-art imaging, single cell analysis and quantitative data analysis. Our **specific lines of research** are aimed to reveal:

- **Novel modulators of stem cell self-renewal and pluripotency**
- **Mechanisms** by which mitochondrial network dynamics and function **regulate early differentiation (i.e. neuronal, cardiac) and human brain development**
- **Ubiquitin-mediated regulation of early differentiation and human brain development**

## *Model systems*



VANDERBILT SCHOOL OF MEDICINE | Basic Sciences

# Alyssa Hasty, PhD

**Cornelius Vanderbilt Professor of Molecular Physiology and Biophysics**
**Associate Dean for Faculty Development**


***alyssa.hasty@vanderbilt.edu***
***615-322-5177***
***https://lab.vanderbilt.edu/hasty-lab***


## Biosketch

**Dr. Alyssa Hasty earned her Ph.D. at Vanderbilt University and completed a postdoctoral fellowship at Tokyo University. She is currently a Professor in the Department of Molecular Physiology and Biophysics and in 2017 she received a Cornelius Vanderbilt Endowed Chair. She was Director of Graduate Studies for the MPB Department for 6 years, was one of the founding members of Women on Track and is currently the Director of the DDRC Career Development program. In 2017, Dr. Hasty was appointed as Associate Dean for Faculty Development of the Basic Sciences in the School of Medicine.**

## Key Publications


**"High CD8 T cell receptor clonality and altered CDR3 properties in adipose tissue of obese mice," 2018, *Diabetes***

**"Links between Immunologic Memory and Metabolic Cycling," 2018, *Journal of Immunology***

**"Obesity impairs adipose tissue macrophage and systemic iron handling," 2014, *Diabetes***

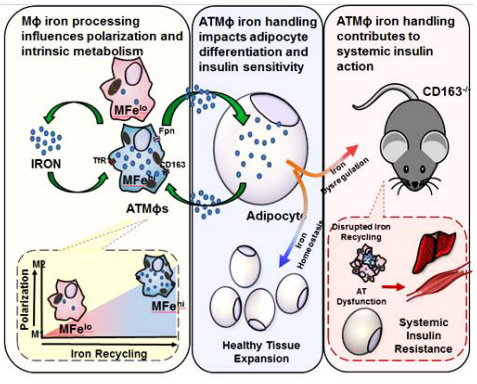

## The Traitor Within: How our immune system can betray us to promote diabetes

We study **how obesity impacts health**. The growing worldwide obesity epidemic is frequently linked to hyperlipidemia, inflammation, and insulin resistance leading to increased risk of diabetes and cardiovascular disease.

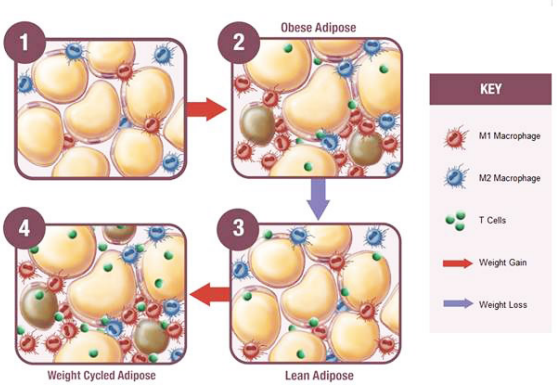
The **long-term goal** of our laboratory is to determine mechanisms by which obesity increases risk for and pathophysiological consequences of these devastating diseases.

**Macrophages** are part of the innate immune system that infiltrate white adipose tissue (fat) in obese rodents and humans, and produce most of the inflammatory cytokines and chemokines secreted from adipose tissue. In addition, their presence has been shown to be temporally associated with the development of insulin resistance.

Our **current research focus** is threefold: to determine mechanisms by which macrophages accumulate in adipose tissue, to determine the role of resident macrophages in normal adipose tissue function, and to determine how other immune cells like eosinophils and T cells also contribute to adipose tissue function.




Macrophage iron handling is critical for adipose tissue health




Adaptive immune cells contribute to worsened metabolic disease in weight cycling

VANDERBILT SCHOOL OF MEDICINE | Basic Sciences

# Emily Hodges, PhD

**Assistant Professor, Department of Biochemistry**
**Vanderbilt Genetics Institute and Vanderbilt Ingram Cancer Center**

***emily.hodges@vanderbilt.edu***
***615-875-9991***
***https://lab.vanderbilt.edu/hodges-lab***

## Biosketch

**Dr. Hodges received her Ph.D. in Functional Genomics from the Karolinska Institute, where she developed high-throughput approaches to investigate the function of newly discovered proteins in mammalian cell culture models. She completed her postdoctoral training at Cold Spring Harbor Laboratory where she pioneered chip-based DNA capture methods that allow rapid targeted sequencing of protein coding genes and ancient DNAs to enable profiling of DNA mutations. Her lab utilizes next-generation DNA sequencing to understand the role of chemical, so-called "epigenetic" DNA modifications in both gene regulation and functional specialization of different blood cell types, and how epigenetic variability can lead to disease susceptibility in immune disorders and cancer.**

## Key Publications

**"De novo DNA demethylation and noncoding transcription define active intergenic regulatory elements," *Genome Research*, Oct 23(10):1601-14, 2013**

**"Directional DNA methylation changes and complex intermediate states accompany lineage specificity in the adult hematopoietic compartment," *Molecular Cell*, 44(1):17-28, 2011**

**"Targeted investigation of the Neanderthal genome by array-based sequence capture," *Science*, 328(5979):723-5, 2010**

## "How epigenetic traits are passed from generation unto generation "

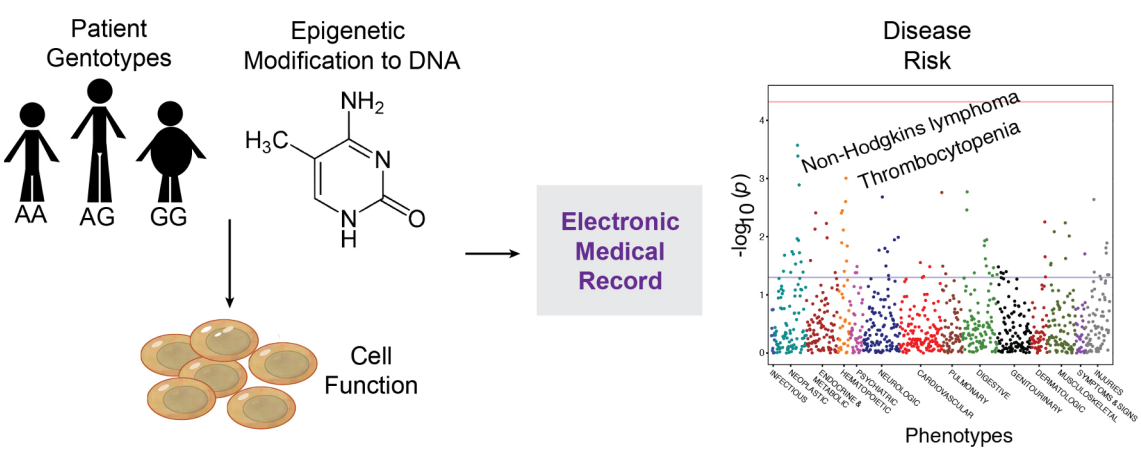
Research in the Hodges Lab **strives to understand how epigenetic features shape human genomes**. We study this relationship on two levels; first, we are interested in **how chemical modifications of DNA, DNA methylation, are established during the specialization of developing cell types**. Second, we are interested in the **relationship between genetics, DNA methylation state (epitype), and how this relationship affects cellular function (phenotype).**

### I. DNA methylation of genomic sequence elements in differentiating cells

Gene regulatory elements called "enhancers" are docking sites for protein-DNA interactions that control gene expression. They are the nodes of complex gene interaction networks that direct cell fate specification and maintain tissue homeostasis. Furthermore, they are believed to be a driving force behind the diversification of organisms. DNA methylation is an important component of this process but **little is understood about how patterns of DNA methylation are established during development. Projects in our lab address these questions utilizing innovative biochemical, functional genomic and bioinformatic approaches.**

### II. Human methylation variation and disease susceptibility

Enhancers display higher DNA methylation variability between species and human individuals than other genomic elements. These differential patterns of enhancer methylation may reflect individual differences in gene regulation and disease susceptibility. **We integrate our understanding of cell-type specific DNA methylation patterns with genetic and phenotype information from the electronic health record to triangulate specific functional relationships between genetic variation and disease risk.**



VANDERBILT SCHOOL OF MEDICINE | Basic Sciences

# Rebecca Ihrie, PhD

**Assistant Professor of Cell & Developmental Biology**
**Assistant Professor of Neurological Surgery**

***rebecca.ihrie@vanderbilt.edu***
***615-936-2951***
***https://my.vanderbilt.edu/ihrielab***

## Biosketch

**Dr. Ihrie earned a B.S. Honors in Biochemistry from the University of Michigan and subsequently received her Ph.D. in Cancer Biology from Stanford University. During her postdoctoral research at the University of California - San Francisco, she studied the stem cells of the young and old brain, including identifying specific signals that change neural stem cells' identity and revealing the architecture of the stem cell niche in pediatric human brain.**

## Key Publications

**"Decreased survival in glioblastomas is specific to contact with the ventricular-subventricular zone, not subgranular zone or corpus callosum," *J Neurooncol*, Apr;132(2):341-349, 2017**

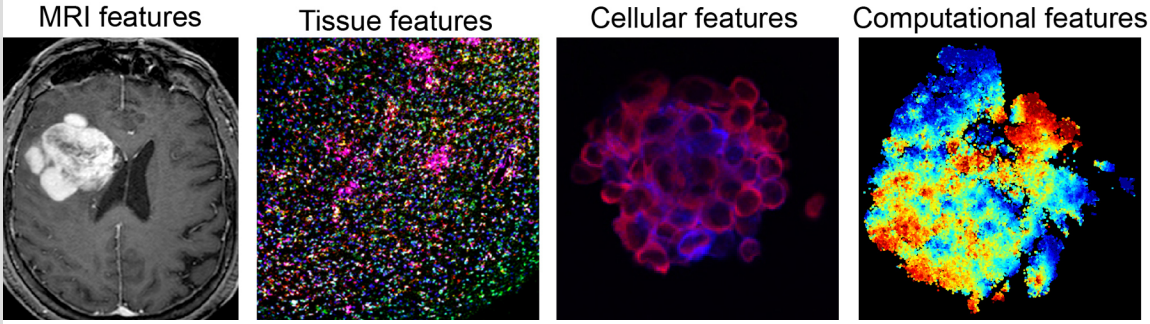
**"Single cell analysis of human tissues and solid tumors with mass cytometry," *Cytometry B Clin Cytom*, Jan;92(1):68-78, 2017**

**"Persistent sonic hedgehog signaling in adult brain determines neural stem cell positional identity," *Neuron*, Jul 28;71(2):250-62, 2011**

## "Understanding normal brain stem cells to target brain tumors"

The Ihrie laboratory is focused on **understanding the connections between stem cells in the brain and brain tumors**. We work with basic scientists, computational biologists, and clinicians to study **pediatric and adult brain tumors**, with the end goal of **finding more successful and targeted treatments**. Current research projects are focused on **tumor development in Tuberous Sclerosis Complex, pediatric gliomas, and adult gliomas including glioblastoma**.

In particular, the lab uses multiple cutting-edge approaches to **map the features of each of millions of individual cells** within brain tumors, allowing us to reveal and **target rare subpopulations of cells** that are **associated with clinically meaningful events like tumor regrowth and resistance to current treatment.** We strive to couple clinical imaging, such as identification of tumor location on MRI, to molecular data on cancer and immune cells, **with the goal of enabling precision medicine approaches in the brain.**



VANDERBILT SCHOOL OF MEDICINE | Basic Sciences

# Jonathan Irish, PhD


**Assistant Professor of Cell and Developmental Biology**
**Assistant Professor of Pathology, Microbiology and Immunology**

***jonathan.irish@vanderbilt.edu***
***615-875-0965***
***https://my.vanderbilt.edu/irishlab***


## Biosketch

**Dr. Irish received his PhD in Cancer Biology from Stanford University where he trained in cancer biology, immunology, and computational biology. He completed postdoctoral training at Stanford in tumor immunology. During this time, he created a new precision medicine approach based on single cell measurements and co-created Cytobank, a cloud computing platform used by thousands of researchers worldwide. His lab now uses artificial intelligence tools to identify and study diseased cells, improve clinical tests, and create new therapies that specifically target rare cells. His group also operates the Cancer & Immunology Core and Mass Cytometry Center of Excellence shared resources.**

## Key Publications


**"Computational immune monitoring reveals abnormal double negative T cells present across human tumor types," *Cancer Immunology Research*, in press, 2018**

**"Discovery of human cell selective effector molecules using single cell multiplexed activity metabolomics," *Nature Communications*, 2;9(1):39, 2018**

**"Characterizing cell subsets in heterogeneous tissues using marker enrichment modeling," *Nature Methods*, 14(3):275-278, 2017**

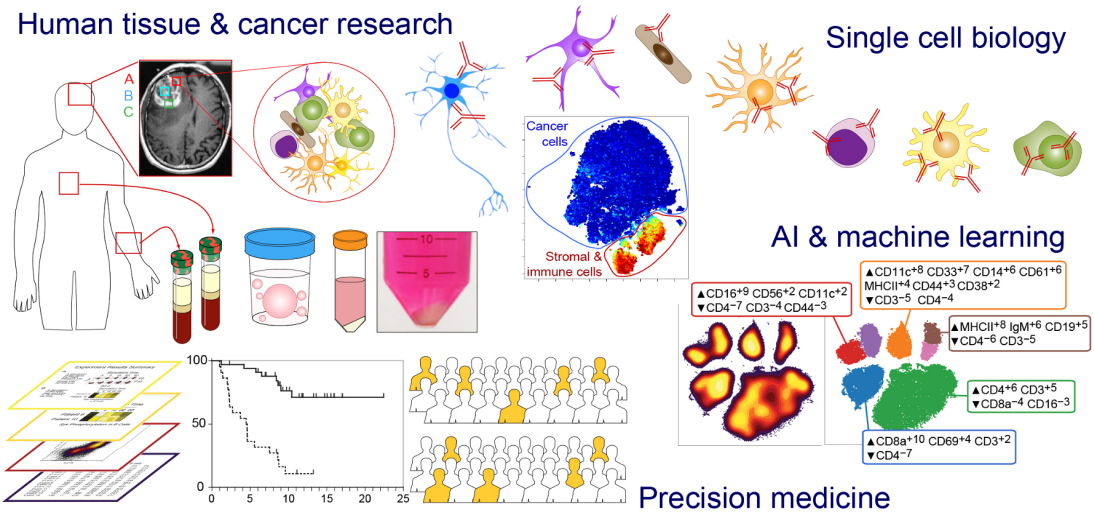

# "Artificial intelligence for human biology: using computers to discover cells & tailor treatments"

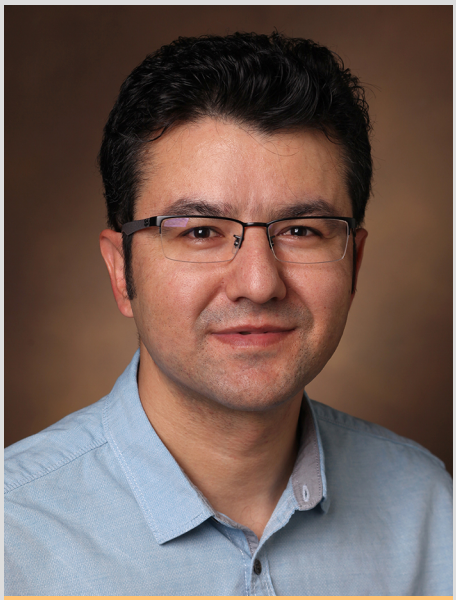
Single cell biology and data science are revolutionizing our understanding of complex, multicellular diseases and therapies. This is especially the case with cancer targeted therapies and immunotherapy, where **an understanding of signaling interactions among cancer cells, microenvironment cells, and immune cells** is critical.

A central goal of our research is to understand **how cell signaling mechanisms govern healthy development and control the outcomes of human diseases and treatments. Our lab creates cutting-edge single cell mass cytometry and phospho-specific flow cytometry tools to search through billions of cells from human tumors, lymph nodes, bone marrow, and blood samples in order to precisely target rare, abnormal cells.**

We are **especially focused on targeting signaling networks and immune interactions in rare cell populations from patient clinical samples** obtained over time during therapy. Our lab members have diverse scientific backgrounds and work at the interface between multiple fields, including computational biology, chemical biology, neuroimmunology, and precision medicine.

We believe great potential exists to **detect diseases earlier and to tailor a patient's therapy to the biological alterations detected in the cells of their disease.** By better understanding biological systems which control development and cell-cell interactions in healthy and diseased contexts, **we can learn to program cells to become therapeutic agents or target malignant signaling events to specifically kill cancer cells.**





VANDERBILT SCHOOL OF MEDICINE | Basic Sciences

# Erkan Karakas, PhD


**Assistant Professor of Molecular Physiology and Biophysics**
**Vanderbilt University School of Medicine**

***erkan.karakas@vanderbilt.edu***
***615-343-4494***
***https://medschool.vanderbilt.edu/mpb/person/erkan-karakas-phd***


## Biosketch

**Dr. Karakas received his PhD in Molecular and Cellular Biology from Stony Brook University where he used X-ray crystallography to study the molecular mechanism of nucleotide excision repair, an essential pathway to protect genome against environmental damage. Prior to joining Vanderbilt University in 2016, he completed his postdoctoral training at Cold Spring Harbor Laboratory, where he solved the structure of N-methyl-D-aspartate (NMDA) receptors, which belong to the ionotropic glutamate receptor family that mediates the majority of excitatory synaptic transmissions in the mammalian brain, at atomic resolution, and investigated how binding of neurotransmitters and drugs regulate receptor activity.**

## Key Publications


**"Activation of NMDA receptors and the mechanism of inhibition by ifenprodil,"** ***Nature*****, 534(7605):63-8, 2016**

**"Crystal structure of a heterotetrameric NMDA receptor ion channel,"** ***Science*****, 344(6187):992-7, 2014**

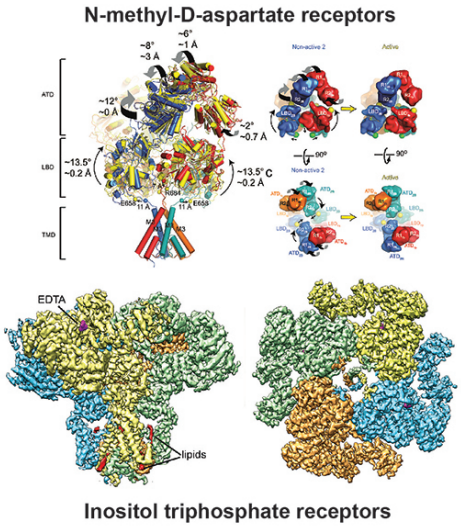
**"Subunit arrangement and phenylethanolamine binding in GluN1/GluN2B NMDA receptors,"** ***Nature*****, 475(7355):249-53, 2011**

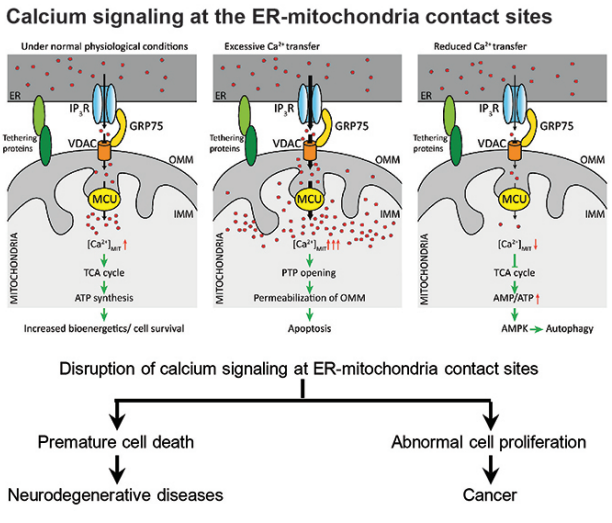

## "Calcium Channels: Structure, function, and pharmacology"

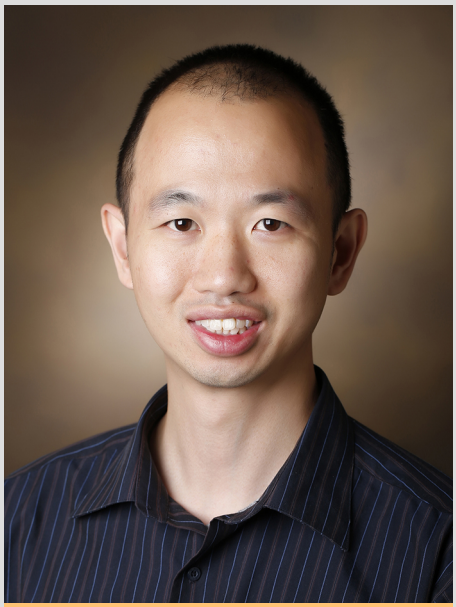
Research in the Karakas Lab focuses on **structural and functional characterization of calcium channels** to elucidate the molecular mechanism of ion channel activity and regulation in healthy and diseased states. Calcium acts as universal messengers required to regulate diverse physiological processes including fertilization, muscle contraction, apoptosis, secretion, and synaptic plasticity. Calcium channels are essential components of the calcium signaling toolkit and their **aberrant activity** is associated with a number of diseases including **cancer, metabolic and neurodegenerative diseases**. Specific targets being studied include:

· ***N*-methyl-D-aspartate receptors (NMDARs)**, ligand-gated calcium channels that belong to the ionotropic glutamate receptor family. NMDARs are involved in synaptic transmission in the central nervous system and essential for basic brain function including learning and memory. Aberrant NMDAR activity is implicated in various neurodegenerative diseases including **Alzheimer's and Parkinson's diseases**, and psychological disorders such as **schizophrenia and depression**.

· **Inter-organelle calcium signaling machinery**, which mediates sustained calcium transfer from endoplasmic reticulum (ER) to mitochondria to maintain cellular bioenergetics. Excessive or reduced calcium transfer leads to **apoptotic cell death or autophagy**, respectively. Consequently, calcium signaling at ER-mitochondria interface plays an essential role in cell fate decisions and could be an **invaluable target**when the cell fate decision machinery is compromised, as observed in **cancer (evasion of apoptosis)** and **neurodegenerative diseases (excessive apoptosis)**.





VANDERBILT SCHOOL OF MEDICINE | Basic Sciences

# Ken Lau, PhD

**Assistant Professor of Cell and Developmental Biology**
**Vanderbilt University School of Medicine**

***ken.s.lau@vanderbilt.edu***
***615-936-6859***
***https://www.mc.vanderbilt.edu/vumcdept/cellbio/laulab***
***@KenLauLab***

## Biosketch

**Ken was born in Hong Kong and grew up in Toronto, Canada. He was the first student in the Proteomics and Bioinformatics graduate program at the University of Toronto. He joined the faculty at Vanderbilt after a successful four-and-a-half-year postdoctoral fellow at MIT and Massachusetts General Hospital, a Harvard teaching hospital. Ken received a Damon Runyon Research Fellowship, as well as an Innovator Award from the American Association of Cancer Research.**

## Key Publications

**"Unsupervised trajectory analysis of single-cell RNA-seq and imaging data reveals alternative tuft cell origins in the gut," *Cell Systems*, 6(1), 37-51, 2018**

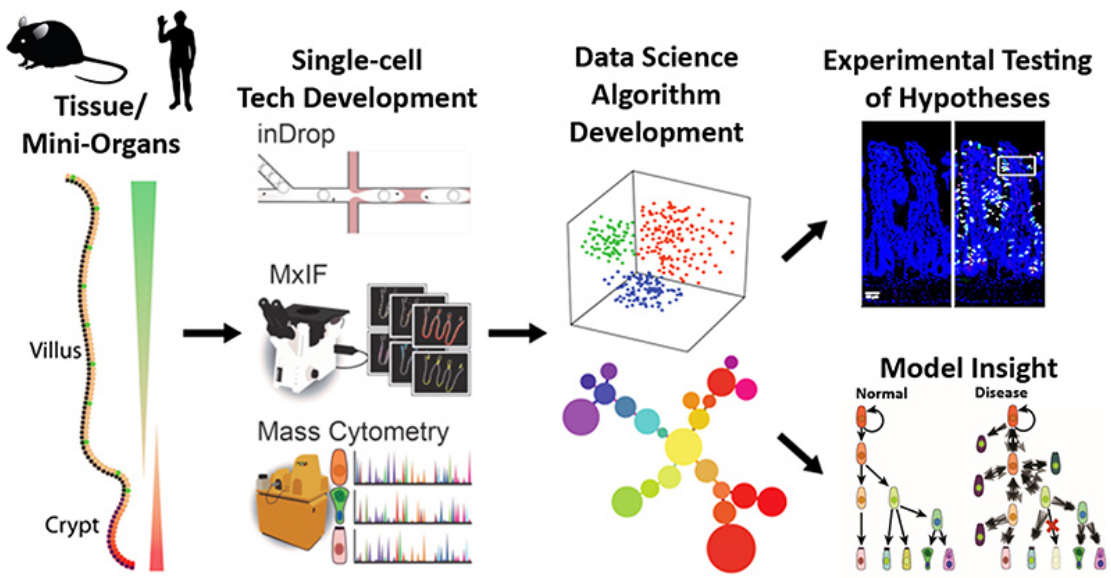
**"Impaired coordination between signaling pathways is revealed in human colorectal cancer using single-cell mass cytometry of archival tissue blocks," *Science Signaling*, 9(449), rs11, 2016**

**"Cytometry-based single cell analysis of intact epithelial signaling reveals MAPK activation divergent from TNF-α-induced apoptosis *in vivo*," *Molecular Systems Biology*, 11(10):835, 2015**

## "Big data modeling of cell-microbe social networks"

The Lau lab considers every one of the 30 plus trillion cells in the body to be unique, thus, **utilizes state-of-the art technologies to profile tissues at the single-cell resolution**. Resulting "big data" consisting of **thousands of data points and dimensions** are **analyzed by data science-driven computational techniques** to determine:

- how altering different cell types in the gut influences the **cell-microbiome ecosystem of Inflammatory Bowel Disease**
- how interactions between the gut microbiome and epithelial cells contribute to **benign colonic polyps progressing to colon cancer**
- how the **origins of colon cancer stem cells** affect the progression of cancer and responses to therapies
- how different **mutations affecting the same pathway** can lead to different outcomes depending on the cellular ecosystem



VANDERBILT SCHOOL OF MEDICINE | Basic Sciences

# Carlos Lopez, PhD


**Assistant Professor of Biochemistry**
**Assistant Professor of Pharmacology**
**Assistant Professor of Biomedical Informatics**

***c.lopez@vanderbilt.edu***
***615-936-1311***
***https://my.vanderbilt.edu/lopezlab***


## Biosketch

**Carlos F. Lopez received his BSc and BLA degrees from University of Miami, his PhD in Physical Chemistry from University of Pennsylvania, and had postdoctoral positions at UT-Austin and Harvard Medical School. His work employs novel theoretical, computational, and numerical tools, in combination with experimental data, to explain and predict cellular dynamic processes. He incorporates Machine Learning, Bayesian Statistics, and Artificial Intelligence methods to link molecular-level interactions with cell-population dynamics processes. He is currently the Vanderbilt University Liaison to Oak Ridge National Laboratory.**

## Key Publications


**"PyDREAM: High-dimensional parameter inference for biological models in Python,"** ***Bioinformatics*****, 34(4), 695-697 (2018)**

**"An unbiased metric of antiproliferative drug effect in vitro,"** ***Nature Methods*****, Vol 13, pp. 497-500 doi:10.1038/nmeth.3852, (2016)**

**"Competition and allostery govern substrate selectivity of cyclooxygenase-2,"** ***Proc. Nat. Acad. Sci. USA*** **,Vol 112, Iss 40, pp. 12366-12371 doi:10.1073/pnas.1507307112 (2015)**


## "From Molecules to Organisms: Numerical methods to explain and predict how cells make good and bad decisions"

The work in the Lopez lab is driven by two overarching goals:

1. Can we **understand the physical and chemical rules that govern cellular processes**?

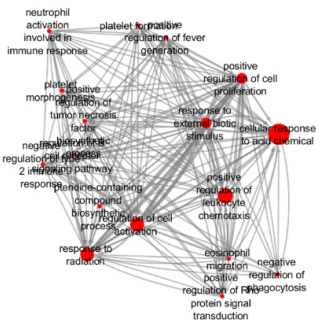
2. Can we **predict how a cell will respond** to a perturbation in health or disease?

Cells must respond to external and internal perturbations such as mutations or toxins. How do cells employ complex biochemical reaction networks to process these intra- and extracellular signals to commit to a given outcome?

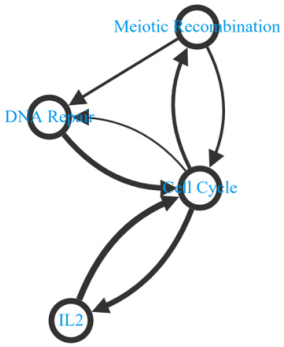
The Lopez lab **employs computational modeling, Machine Learning, and dynamic network analysis methods** to **explain and predict cell behaviors in health and disease**. A central goal in the lab is to **understand the molecular mechanisms that drive cancer cells to respond to treatments or avoid treatment and seed drug resistance and cancer relapse**.

Despite the immediate significance of this work to cancer, a fundamental understanding of cell-decision processes will be generalizable to other areas of biology, including **drug development and bioengineering applications**.

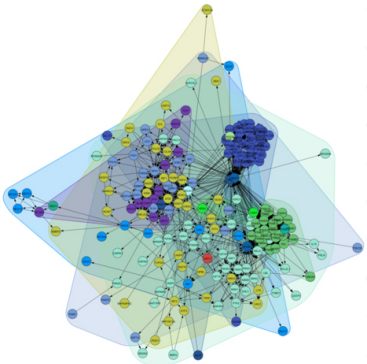
Combination drugs mechanism of action




Cisplatin Mechanism of action




Misoprostol mechanism of action



VANDERBILT SCHOOL OF MEDICINE | Basic Sciences

# Jason MacGurn, PhD

**Assistant Professor of Cell and Developmental Biology**
**Vanderbilt University School of Medicine**

***jason.a.macgurn@vanderbilt.edu***
***615-343-4259***
***https://lab.vanderbilt.edu/macgurn-lab***

## Biosketch

**Jason obtained his undergraduate degree at The University of Chicago and received his PhD in Biochemistry and Biophysics from UCSF. He conducted postdoctoral research at Cornell University using biochemical and imaging approaches to study how cellular proteins are targeted for degradation. In 2013, Jason started his lab in the Department of Cell and Developmental Biology at Vanderbilt to investigate mechanisms of cellular protein degradation in the context of human disease. Jason has been the recipient of the Blavatnik Award for Young Scientists, the NIH Pathway to Independence Award, and a Junior Investigator Award from the American Federation for Aging Research.**

## Key Publications

**"Ubiquitin turnover and endocytic trafficking in yeast are regulated by Ser57 phosphorylation of ubiquitin," *eLife*, 2017, e29176**

**"COPI mediates recycling of an exocytic SNARE by recognition of a ubiquitin sorting signal," *eLife*, 2017, e28342**

**"TORC1 Regulates Endocytosis via Npr1-mediated Phosophinhibition of a Ubiquitin Ligase Adaptor," *Cell*, 2011, 147(5): 1104-17**

## "Seeking a healthy balance among the many paths leading to protein destruction"

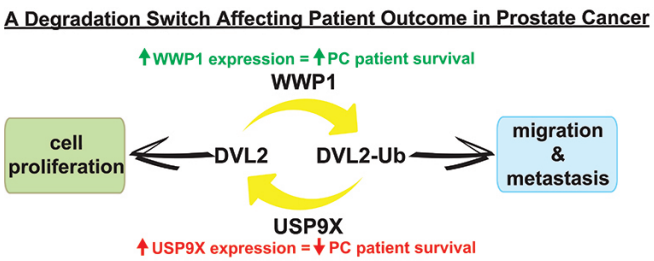
The main objective of the MacGurn Lab is to **dissect cellular mechanisms of protein degradation** and ultimately to leverage this knowledge towards the **development of strategies to fight human disease**.

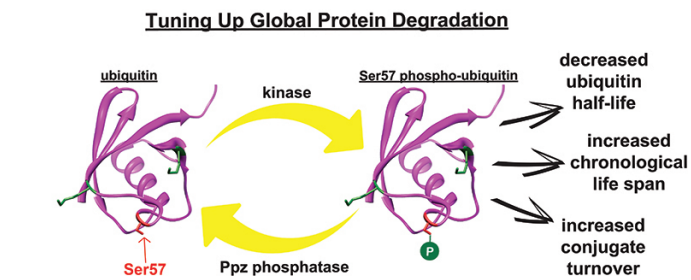
**Objective #1: Harness protein degradation pathways to fight cancer.**

In the MacGurn Lab, we are learning how to **manipulate protein degradation machinery to target destruction of cancer-driving proteins**. This has led to the identification of novel chemical strategies for **inhibition of important signaling pathways that promote cancer progression**. For example, we have identified one protein degradation switch that we are currently exploring as a possible therapeutic target for **treatment and prevention of advanced forms of prostate cancer**.

**Objective #2: Develop strategies for "tuning up" global protein degradation.**

In the course of aging, and particularly in neurodegenerative states like **Alzheimer's disease and Parkinson's disease**, our cells suffer a dramatic decline in protein degradation capacity. Furthermore, there is an emerging consensus that restoring degradation capacity can reverse cellular pathologies associated with neurodegeneration and aging. **We have discovered a novel mechanism for "tuning up" global protein degradation in eukaryotic cells, and we are actively investigating how this affects aging in eukaryotic cells.**



VANDERBILT SCHOOL OF MEDICINE | Basic Sciences

# Gregor Neuert, PhD


**Assistant Professor of Molecular Physiology and Biophysics**
**Assistant Professor of Pharmacology**
**Assistant Professor of Biomedical Engineering**

***gregor.neuert@vanderbilt.edu***
***615-343-6404***
***https://lab.vanderbilt.edu/neuert-lab***


## "Understanding how an individual cell perceives and responds to its environment in healthy and disease conditions"

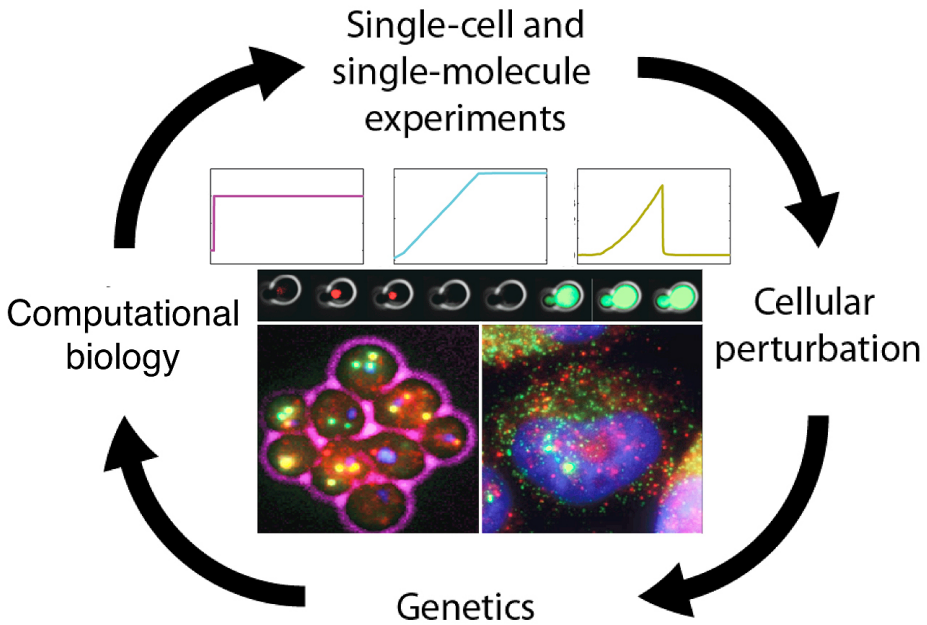
The Neuert lab combines quantitative single cell and single molecule experiments with dynamic cellular perturbations, genetics and computational biology to **explore the fundamental mechanism that enable cells to perceive and respond to physiologically relevant environmental changes**. Specific questions of interest are:

How do individual **cells perceive physiologically relevant environments**?

How do **proteins generate dynamic behavior** within a single cell?

How do **cells regulate genes** in physiologically relevant dynamic and stochastic environments?

How to computationally model and predict single cell behavior to gain novel biological insight?




## Biosketch

**Gregor was born in Germany and educated interdisciplinary through a master degree in engineering from TU-Ilmenau in Germany, a Ph.D. in physics from LMU Munich, and postdoctoral research in biology at MIT through a Deutsche Forschungs Gemeinschaft fellowship. He joined the Vanderbilt faculty in 2012, received an NIH New Innovator Award, and is a member of the founding executive committee of the Vanderbilt Data Science Institute.**

## Key Publications


**"Distribution Shapes Govern the Discovery of Predictive Models for Gene Regulation", *Proceedings of the National Academy of Sciences*, 115(29):7533-7538, 2018**

**"Finite state projection based bounds to compare chemical master equation models using single-cell data", *Journal of Chemical Physics*, 145(7):074101, 2016**

**"Integrating single-molecule experiments and discrete stochastic models to understand heterogeneous gene transcription dynamics", *Methods*, 85:12-21, 2015**

VANDERBILT SCHOOL OF MEDICINE | Basic Sciences

# Adrian Olivares, PhD


**Assistant Professor of Biochemistry**
**School of Medicine**

***adrian.olivares@vanderbilt.edu***
***615-322-4539***
***https://lab.vanderbilt.edu/olivares-lab***


## Biosketch

**Dr. Olivares received his PhD in Molecular Biophysics and Biochemistry from Yale University where he studied how dimeric myosin motor proteins coordinate their biochemical cycles to move along actin filaments. He then completed his postdoctoral training at the Massachusetts Institute of Technology, where he used single molecule force spectroscopic techniques to probe how energy-dependent proteases mechanically unfold proteins during substrate degradation. Work in his lab focuses on creating new tools to visualize and manipulate force-sensing and force-producing machines within the cell and better understand the interplay between the physical and biochemical microscopic world.**

## Key Publications


**"Effect of directional pulling on mechanical protein degradation by ATP-dependent proteolytic machines,"** ***Proceedings of the National Academy of Sciences U. S. A.*****, 114(31):E6306-13, 2017**

**"Mechanochemical basis of protein degradation by a double-ring AAA+ machine,"** ***Nature Structural & Molecular Biology*****, 21(10):871-5, 2014**

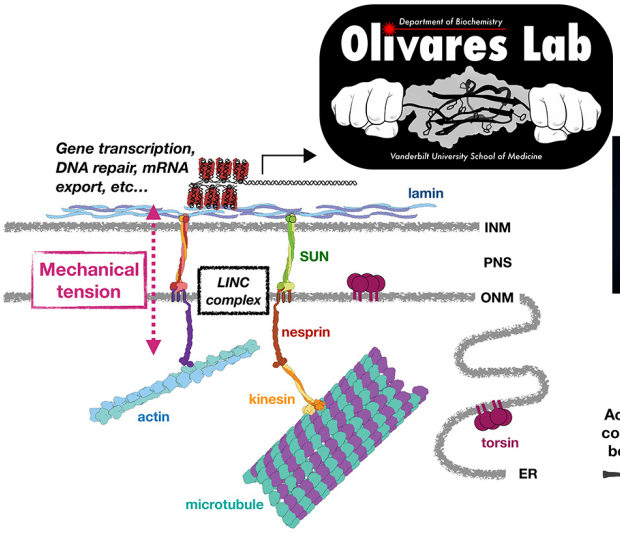
**"Single-molecule protein unfolding and translocation by an ATP-fueled proteolytic machine,"** ***Cell*****, 145(2):257-67, 2011**

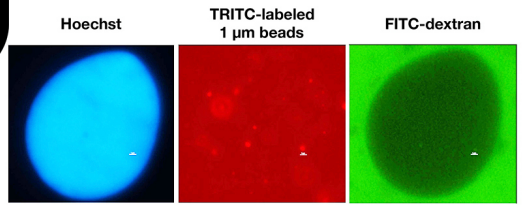

## "Probing how the cell nucleus senses and responds to mechanical forces, one molecule at a time"

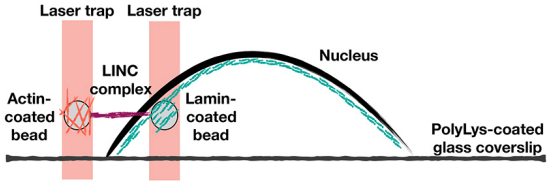
Two fundamental questions my lab aims to address are: how does the **cell use mechanical forces to drive biochemical processes across lipid bilayers**, and how does the **nucleus sense and transform mechanical force into biochemically meaningful signals that influence cellular development and function**? Much evidence suggests that the nucleus can directly sense physical forces through protein complexes spanning the nuclear envelope (NE), though the molecular mechanisms of nuclear force sensing are not well understood.

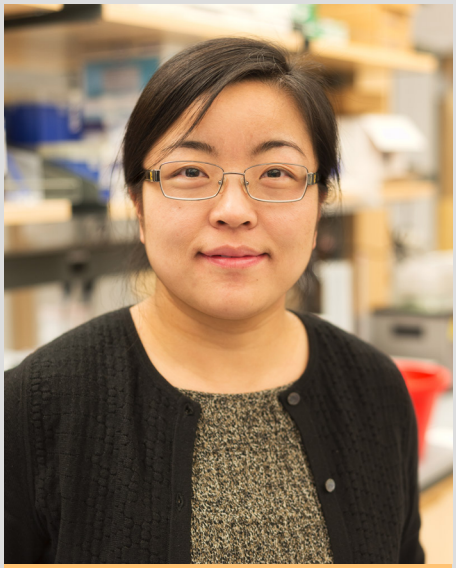
Mutations or dysfunction within this network of membrane embedded and associated proteins lead to changes in NE architecture and to human disease including neurological disorders, cardiomyopathy, muscular dystrophy, and cancer. Though much work has been done to characterize how cell adhesion molecules and the underlying cytoskeleton sense and convert mechanical force into biochemically meaningful reactions, **a mechanistic understanding of nuclear mechanical signaling is lacking**.

Using a combination of single-molecule force spectroscopy, biochemical reconstitution of NE components responsible for mechanical signaling, and biophysical methods, **we hope to unravel the molecular details governing mechanical signaling at the nucleus.**





VANDERBILT SCHOOL OF MEDICINE | Basic Sciences

# Yi Ren, PhD

**Assistant Professor of Biochemistry**
**Vanderbilt University School of Medicine**

***yi.ren@vanderbilt.edu***
***615-343-2914***
***https://lab.vanderbilt.edu/ren-lab***

## Biosketch

**Yi Ren grew up in China and became interested in science at an early age. Her father, a chemical engineer, had set up a home laboratory where Yi spent time watching and playing after school. She went to Fudan University in Shanghai for college. She then came to the US and received her PhD from Princeton University, followed by postdoctoral training with late Nobel laureate Dr. Günter Blobel at the Rockefeller University. Yi joined the faculty at Vanderbilt in 2016. Work in her lab centers on the process of nuclear mRNA export, which is critical for cellular life and is exploited by diverse viruses to counteract host defenses.**

## Key Publications

**"Structural Basis for Influenza Virus NS1 Protein Block of mRNA Nuclear Export" Manuscript submitted for publication.**

**"Structural and Biochemical Analysis of the DEAD-box ATPase Sub2 in Association with THO or Yra1" *Elife* 2017, e20070**

**"Vesiculoviral Matrix (M) Protein Occupies Nucleic Acid Binding Site at Nucleoporin Pair (Rae1•Nup98)" *PNAS* 2014, 111(25):9127-32**

## Evading the Invaders: Reverting unhealthy nuclear imprisonment of human RNA by viruses

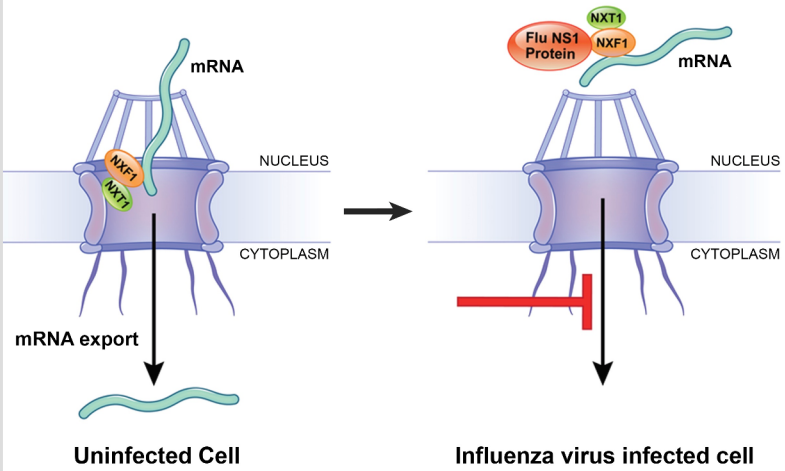
All cellular life relies on the integrity of gene expression. Human cells carry more than 20, 000 different mRNAs. Fully processed mRNAs in the nucleus are exported to the cytoplasm where protein translation occurs. This **nuclear mRNA export process** is exploited by a large number of **viruses to block host gene expression as a strategy to counteract host defenses**.

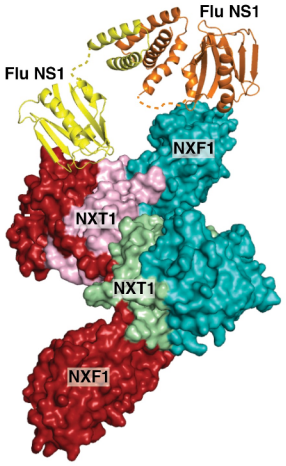
The Ren lab combines the power of biochemistry, structural biology, and cell biology to **elucidate the molecular details of viral strategies targeting nuclear mRNA export**.

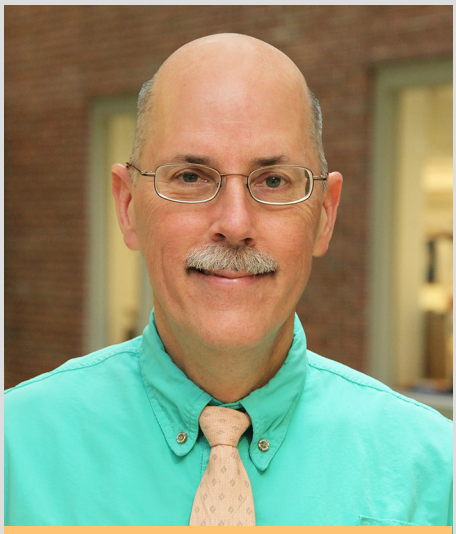
- **Influenza A Virus (IAV)**
  Influenza virus remains a major public health threat killing ~250,000-500,000 people yearly. Specific questions being studied include: **What is the cellular target of IAV**? How does **IAV inhibit nuclear mRNA export**? Which **mRNAs are retained in the host nucleus** upon IAV infection? How does **blockage of nuclear mRNA export** contribute to inhibition of host immunity?

- **Vesicular Stomatitis Virus (VSV)**
  VSV blocks host mRNA export and limits expression of interferons, a family of signaling proteins that activate host defense systems. **We seek to understand the molecular underpinnings of VSV mediated host mRNA export blockage.**

Our work brings conceptual novelty to the fields of virology, immunology, and cell biology of nuclear transport. **Results** generated from these studies will provide significant implications for **developing therapeutic interventions** aiming to revert nuclear imprisonment of host mRNAs by viruses.





VANDERBILT SCHOOL OF MEDICINE | Basic Sciences

# Charles (Chuck) R. Sanders, PhD


**Professor of Biochemistry**
**Associate Dean for Research**
**Aileen M Lange and Annie Mary Lyle Chair in Cardiovascular Research**
**Professor of Medicine**

***chuck.sanders@vanderbilt.edu***
***615-936-3756***
***http://structbio.vanderbilt.edu/sanders***


## Biosketch

**Chuck was born in Columbus, Ohio and did his undergraduate work at Milligan College, TN, followed by his Ph.D at The Ohio State University and an NIH postdoctoral fellowship at Yale University. He joined the faculty at Vanderbilt in 2002 following a decade on the faculty at Case Western Reserve University. Among other honors he is the recipient of the 2013 Hans Neurath Award of the Protein Society and the 2012 Anatrace Membrane Protein Award of the Biophysical Society. He served 13 years as an Associate Editor and then Acting Editor-in-Chief of the ACS journal *Biochemistry*.**

## Key Publications


**"Mechanisms of KCNQ1 Channel Dysfunction in Long QT Syndrome Involving Voltage Sensor Domain Mutations," *Science Advances,* 4, eaar2631, 2018**

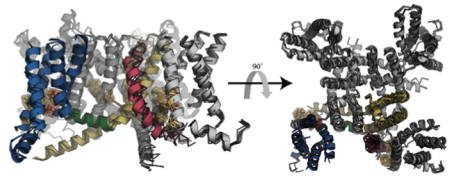
**"The Amyloid Precursor Protein has a Flexible Transmembrane Domain and Binds Cholesterol," *Science,* 336, 1168-1171, 2012**

**"Implications of the differing roles of the β1 and β3 transmembrane and cytoplasmic domains for mammalian integrin function," *eLife* , e18633, 2016**

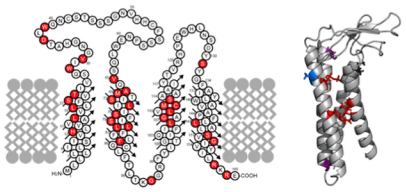

## "How do human membrane proteins contribute to various diseases?"

The Sanders lab uses the tools of **Biochemistry, Structural Biology**, and **Chemical Biology** to explore the roles of membrane proteins in human diseases. Specific questions being addressed include:

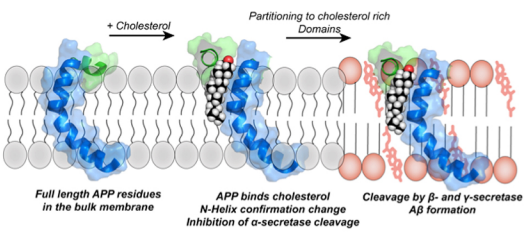
- How does the binding of cholesterol to the **amyloid precursor protein** promote **Alzheimer's disease**?
- How does a healthy **TREM2** receptor help suppress **neuroinflammation** that otherwise might accelerate **Alzheimer's, Parkinson's**, and other neurodegenerative diseases.
- Can folding defect in human **peripheral myelin protein 22** be suppressed with pharmacological chaperones as a route to treating the common peripheral neuropathy, **Charcot-Marie-Tooth disease**?
- How do the more than 600 known mutations in the **KCNQ1 potassium channel** act to alter the function of this channel, resulting in the **long-QT syndrome (LQTS) cardiac arrhythmia** and associated sudden death? Can this information be leveraged to help predict patient predisposition to LQTS for preventive personalized/precision medicine?
- What roles do cell surface **integrin receptors** play in promoting fibrosis in **chronic kidney disease**, the most common killer of patients with **type II diabetes**?



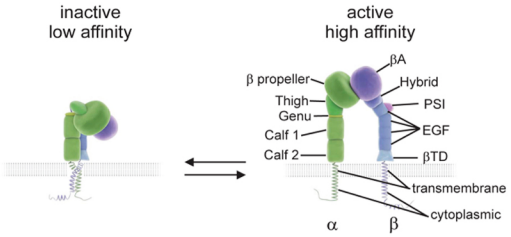
**How Defects in the KCNQ1 Potassium Channel Cause Long QT Syndrome Cardiac Arrhythmia**



**How Mutations in Peripheral Myelin Protein 22 Cause Charcot-Marie Tooth Disease**




**How Cholesterol Promotes Alzheimer's Disease**




**How Integrin Receptors Link Cells To Surrounding Tissue in Health and Kidney Disease**

VANDERBILT SCHOOL OF MEDICINE | Basic Sciences

# Bryan Venters, PhD


**Assistant Professor of Molecular Physiology and Biophysics**
**Vanderbilt University School of Medicine**

***bryan.venters@vanderbilt.edu***
***615-322-4761***
***https://lab.vanderbilt.edu/venters-lab***


## Biosketch

**Dr. Venters earned his PhD in Molecular Biology from Pennsylvania State University where he studied epigenetic and gene regulatory mechanisms. During his postdoctoral training at Penn State, he developed state-of-the-art functional genomic tools that enable examination of protein-DNA interactions with the highest resolution currently possible. Research in the Venters lab involves (1) development of cutting edge functional genomic and computational tools, (2) application of these tools to dissect fundamental erythroid cell biology, and (3) translation of key discoveries into new therapeutics and treatment strategies.**

## Key Publications


**"Integrative view of epigenetics in erythroid cells," 2018, *Current Opinion in Hematology*, 25(3):189-195.**

**"Epo reprograms the epigenome of erythroid cells," 2017, *Experimental Hematology*, S0301-472X(17)30133-9.**

**"Genomic Organization of Human Transcription Initiation Complexes," 2016, *PLoS ONE*, 11(2):e0149339.**


## "How hormones impact red blood cells in health and disease"

The Venters lab leverages their expertise in Molecular Biology, Functional Genomics, and Bioinformatics to **study the molecular mechanisms underlying hematological diseases**. Our research is guided by two overarching questions:

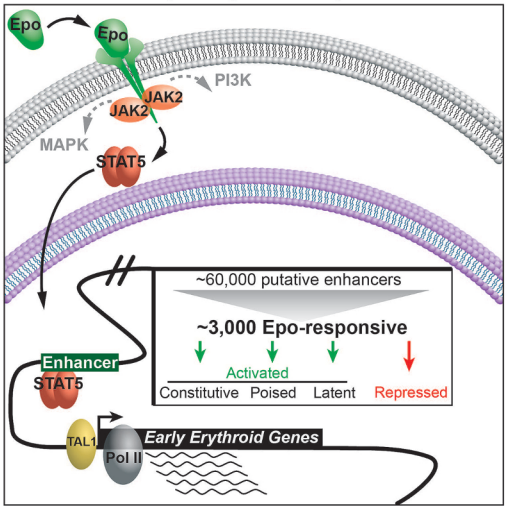
**1. How does EPO signaling control RNA polymerase II kinetics during red cell development?**

**2. How are early EPO-responsive genes regulated by transcription factors and enhancers?**

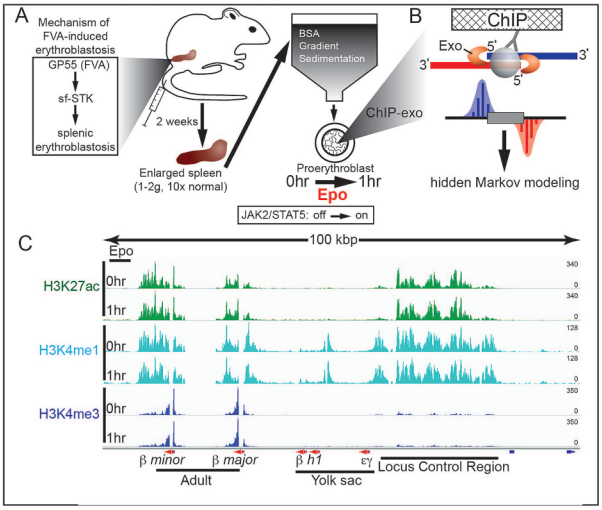
**Erythropoietin (EPO)** is the primary hormone regulator that controls erythroid cell maturation, a process that is required for the **daily replenishment of nearly 1% (200 billion) of the circulating red blood cells**. Recombinant human EPO (trade name Epogen) is a $5 billion/year drug that is used to treat chronic anemia. However, the therapeutic use of EPO has been controversial since reports have emerged of its link to **increased risk of heart attacks, tumor growth, and death in some cases**. An understanding of EPO function has advanced substantially, but **certain aspects of the EPO signaling pathway remain unknown. In particular, how EPO signaling controls erythroid expression patterns through epigenetic and transcriptional mechanisms remains poorly understood.**

Thus, the overarching goal of our research is to **understand the gene regulatory mechanisms governing EPO-induced erythroid differentiation, and translating these discoveries into potential therapeutic interventions.**

Overview of the pathways studied by the Venters lab




EPO reprograms the epigenome of mouse erythroid cells



VANDERBILT SCHOOL OF MEDICINE | Basic Sciences

# Marija Zanic, PhD


**Assistant Professor of Cell and Developmental Biology**
**Assistant Professor of Chemical and Biomolecular Engineering**
**Assistant Professor of Biochemistry**

***marija.zanic@vanderbilt.edu***
***615-343-8501***
***https://lab.vanderbilt.edu/marija-zanic-lab***


## Biosketch

**Dr. Zanic received her PhD in Physics from the University of Texas at Austin, followed by postdoctoral training at the Max Planck Institute of Molecular Cell Biology and Genetics in Dresden, Germany. She spent a year as an Associate Research Scientist at Yale University prior to starting her independent laboratory at Vanderbilt in 2014. The Zanic laboratory combines the tools of biology and physics to elucidate the fundamental mechanisms underlying dynamic intracellular architecture. Zanic is a recipient of the Career Development Award from the Human Frontier Science Program, the Maximizing Investigators' Research Award from the NIH, and the Searle Scholars Award.**

## Key Publications


**"Microtubule minus-end aster organization is driven by processive HSET-tubulin clusters," *Nature Communications*, 9:2659, 2018**

**"Human CLASP2 specifically regulates microtubule catastrophe and rescue," *Molecular Biology of the Cell*, 29(10):1168-1177, 2018**

**"Synergy between XMAP215 and EB1 Increases Microtubule Growth Rates to Physiological Levels," *Nature Cell Biology*, 15(6):688-93, 2013**


## "Building the cell's skeleton to understand how cells change shape, move, and divide"

A look inside of every living cell reveals a world of dynamic intracellular structures. One of the core cellular building blocks are **microtubule polymers**, vital for processes such as **cell division, intracellular transport and neuronal development**. Not surprisingly, **misregulation of the microtubule network causes human disease**, including many types of cancers, as well as neurological disorders.

**Our research aims to discover the molecular mechanisms that drive dynamic remodeling of the microtubule network architecture**, essential for its proper cellular function. What are the molecular rules that govern whether a microtubule grows or shrinks at any given moment in time? What are the mechanisms used by the microtubule-associated proteins that regulate microtubule behavior? How does this complex network of regulators collectively orchestrate dynamic remodeling of the microtubule cytoskeleton in vastly diverse cellular contexts? To address these questions we take an interdisciplinary approach, **combining molecular and cell biology, biochemistry, engineering and physics**.

Uniting the tools of many disciplines, our work aims to **provide a fundamental understanding of how cells engineer large-scale, dynamic structures essential for life**. Understanding of the underlying mechanisms will allow us to manipulate dynamic intracellular architectures, ultimately facilitating new, better strategies to fight human disease.