



**New York Consortium for  
Interdisciplinary Training in  
Kidney, Urological & Hematological  
Research**

**Summer Undergraduate Research Internship Program**

Laboratories in the **NYC Train KUHR** program that are accepting applications for the **2026 Summer Undergraduate Research Internship** at the Albert Einstein College of Medicine, Columbia University Irving Medical Center, Icahn School of Medicine at Mount Sinai, and Renaissance School of Medicine at Stony Brook University.

**Albert Einstein College of Medicine**

**Michael J. Ross, MD**

Professor

Department of Medicine (Nephrology)

Department of Developmental & Molecular Biology,

Chief, Department of Medicine Division of Nephrology

**Research Interest:** The major research focus of the Ross laboratory is to identify novel mechanisms involved in renal epithelial and podocyte injury, HIV-related kidney diseases, and Apolipoprotein L1 (APOL1) nephropathies. His laboratory uses in vitro approaches and murine models to investigate these mechanisms and generate new strategies to prevent and treat kidney diseases.

**Michal Melamed, MD, MHS**

Adjunct Clinical Professor

Department of Medicine (Nephrology)

Department of Pediatrics (Pediatric Nephrology)

**Research Interest:** The focus of Dr. Melamed's clinical research is in the epidemiology and clinical trials of kidney disease progression, health equity and complications of kidney disease. She is investigating the effects of potassium citrate on bone strength in kidney disease, innovative care models for kidney transplant listing and cardiovascular effects of treatment of metabolic acidosis in kidney disease.

**Matthew Abramowitz, MD, MS**

Professor

Department of Medicine (Nephrology)

**Research Interest:** Dr. Abramowitz's research focuses on metabolic dysregulation in patients with chronic kidney disease and its effects on skeletal muscle homeostasis and physical function. Our patient-oriented research employs physical function testing, novel MRI methodologies, and histologic, transcriptomic, and proteomic characterization of human skeletal muscle obtained from patient biopsies.

**Kimberly J. Reidy, M.D**

Associate Professor,

Department of Pediatrics (Pediatric Nephrology)

Chief, Division of Pediatric Nephrology

**Research Interest:** The main focus of the Reidy laboratory is on how Par1a/b developmental signaling pathways modify healthy and maladaptive kidney repair after podocyte and kidney tubular injury. In addition, Dr. Reidy is involved in clinical and translational research studies investigating effects of *APOL1* risk variants on preeclampsia and perinatal outcomes, preterm birth effects on podocyte development and risk for adverse glomerular disease outcomes, and clinical trials in pediatric chronic kidney disease.

**Wei Chen, MD, MSc**

Associate Professor

Department of Medicine (Nephrology)

Department of Developmental & Molecular Biology

**Research Interest:** In patients with CKD, arterial calcification is common, with a prevalence approaching 80%, and contributes to high cardiovascular mortality. The long-term goal of our research program is to understand the pathophysiology of arterial calcification and to develop new treatment strategies for it. Using bench-to-bedside translational approaches, we study the role of calciprotein particles, atypical FAT1 cadherin, and cellular interaction in the development of arterial calcification in people with CKD. We characterize arterial calcification in people with CKD using the state-of-the-art <sup>18</sup>F-NaF PET/CT. We use both in vitro and in vivo murine models.

**Kelvin P. Davies, PhD**

Professor

Department of Urology

Department of Molecular Pharmacology

**Research Interest:** Our laboratory employs molecular and biochemical techniques to gain deeper insights into the fundamental mechanisms underlying benign urogenital diseases, with a primary focus on erectile and bladder dysfunction. Our major research interests currently are centered on, i) Investigating cavernous nerve regeneration following radical prostatectomy, ii) Determining the pivotal role of potassium channels in the physiology of both erectile and bladder functions and iii) Exploring the application of nanotechnology for the treatment of urogenital diseases. Overall, our research endeavors aim to advance the understanding and treatment of urogenital disorders.

**Sylvia O. Suadicaní, PhD**

Professor

Department of Urology

Department of Molecular Pharmacology

**Research Interest:** The major research focus of the Suadicaní laboratory is to investigate mechanisms underlying the development of urogenital conditions to identify novel molecular targets to prevent and treat these conditions. Current studies are addressing bladder dysfunction in diabetes and aging, interstitial cystitis and chronic pelvic pain, and female sexual dysfunction. Her laboratory uses both animal and cellular models combined with physiological, behavioral, pharmacological, molecular, and histological approaches to investigate these mechanisms. The Suadicaní laboratory is currently seeking collaborations to implement the use of nanotechnology to improve and devise novel delivery systems for intravesical, bladder targeted drug treatment and gene therapy.

**Rebeca San Martín, PhD**

Assistant Professor

Department of Oncology (Medical Oncology)

Department of Cell Biology

**Research Interest:** The research focus of the San Martín lab is to define the mechanisms by which wound repair-like responses contribute to diseases of the prostate, including benign prostatic hyperplasia (BPH), prostate cancer, and prostate cancer bone metastasis. We are particularly interested in how contact with these microenvironments induce changes in genome organization as a mechanism of disease progression.

**Eric E. Bouhassira, Ph.D.**

Professor

Department of Cell Biology

Department of Medicine (Oncology & Hematology)

Department of Oncology (Hematology)

Ingeborg and Ira Leon Rennert Professor of Stem Cell Biology and Regenerative Medicine

**Research Interest:** Dr. Bouhassira's research focuses on developing blood-forming (hematopoietic) stem cells that can differentiate into red blood cells, T cells, platelets, and all other cell types that comprise blood. This work could potentially aid patients needing transfusions and also save lives by expanding the immunology diversity of hematopoietic stem cells available for transplant. His lab is also interested in epigenetic regulation in the erythroid and hematopoietic lineage with a focus on DNA replication and DNA methylation.

**B. Hilda Ye, Ph.D.**

Associate Professor

Department of Cell Biology

**Research Interest:** Research in Dr. Ye's lab focuses on transcriptional regulation and cell signaling control of normal lymphocyte development and lymphoid malignancies using models for adult T-cell leukemia/lymphoma (ATLL), a disease of malignant CD4+ T cells that develops in 4-5% of individuals infected with the human T-cell lymphotropic virus 1 (HTLV-1). Ongoing studies support the concept that North American- and Japanese-ATLL share similarities in clinical behavior and pathogenesis but also have clear distinctions in mutation pattern, cell cycle regulation, immunophenotype, and response to experimental therapeutics. Since host anti-HTLV-1/anti-ATLL immune response plays a key role in ATLL development, her lab is also examining how host immunity, in particular CD8+ cytotoxic response, is impacted during ATLL development. These studies will uncover the way for novel pathway-targeted and immune-modulatory therapies for ATLL patients and possibly other types of T-cell malignancies.

**Aditi Shastri, M.B.B.S.**

Associate Professor

Department of Oncology (Medical Oncology)

Department of Medicine (Oncology & Hematology)

Department of Developmental & Molecular Biology

**Research Interest:** Dr. Shastri's lab has a strong interest in exploring novel treatment approaches for myelodysplastic syndromes (MDS) and acute myeloid leukemia (AML) after the failure of therapy with hypomethylating agents. Her lab is specifically focused on therapeutically targeting the transcription factor STAT3 in MDS and AML. In addition, Dr. Shastri is an advocate for addressing health disparities across the spectrum of cancer care and engages in health disparities research with the use of large clinical datasets such as the SEER and SEER-Medicare cancer registries.

**David C. Spray, PhD**

Professor

Dominick P. Purpura Department of Neuroscience

Department of Medicine (Cardiology)

**Research Interest:** Research in the Spray laboratory is centered on physiological and cell/molecular biological studies of gap junctions, the intercellular channels that allow cells to directly exchange ions and metabolites. Major projects of the laboratory are attempting to resolve the role of gap junctions and extracellular signaling in (1) pain, involving mouse models of orofacial and hindlimb pain and of chronic pelvic pain (in collaboration with the Suadicani lab), in (2) astrocyte mechanosensitivity and interaction with extracellular matrix in control of blood-

brain-barrier and cortical lamination in a mouse model of a collagen IV mutation, and in (3) bone mechanotransduction (with the Thi Lab and members of the Biomedical Engineering Department, CCNY). These studies utilize a variety of preparations, including primary cultures of cells from transgenic mice with altered expression of connexin and other genes and transfection of wildtype and mutated connexin sequences into communication deficient cell lines. Techniques include intracellular recordings with conventional and ion-selective microelectrodes, photomanipulation such as FRAP, optical monitoring of intracellular ionic activities (especially  $\text{Ca}^{2+}$  and propagated  $\text{Ca}^{2+}$  waves), patch clamp recording of single channels and whole cell currents and standard molecular biological and immunological methods such as Northern and Western blot analyses, confocal immunostaining and RT-PCR.

**Mia M. Thi, PhD**

Associate Professor.

Department of Orthopaedic Surgery.

Department of Molecular Pharmacology.

**Research Interest:** The primary focus of the Thi laboratory is to understand the molecular and cellular mechanisms involved in how cells sense, transduce and signal mechanical stimuli and how cells work in synchrony to propagate locally generated signals throughout the skeletal tissue and others mechanosensitive tissues such as endothelium, urothelium by means of receptor, junctional, cytoskeletal, and focal adhesion proteins under healthy and pathological conditions. Her laboratory uses both in vivo and in vitro approaches to interrogate these mechanisms.

**Columbia University Irving Medical Center**

**Fangming Lin, MD. PhD**

Rustin McIntosh Professor of Pediatrics

Director, Division of Pediatric Nephrology and Hypertension

Columbia University Medical Center

**Research Interest:** The Lin laboratory studies pathobiology of acute kidney injury (AKI) and chronic kidney disease (CKD) with a focus on molecular basis of cell stress, injury and repair. Dr. Lin also treats a growing population of humans who have high risk of developing kidney disease and hypertension due to premature birth and low nephron endowment. Her research team has generated new mouse models of congenital nephron deficits and is using these animal models to understand cellular and molecular mechanisms of renal and cardiovascular disease in humans born preterm.

**Icahn School of Medicine at Mount Sinai**

**Lisa M. Satlin, MD**

Professor & System Chair of Pediatrics (Nephrology)

Department of Medicine (Nephrology)

**Research Interest:** The focus of the Satlin laboratory is on defining the mechanisms leading to the acquisition, maintenance, and regulation of transepithelial transport in the mammalian cortical collecting duct, a nephron segment responsible in the adult for the final renal regulation of total body  $\text{K}^+$  and  $\text{Na}^+$  homeostasis. Specifically, her lab continues to expand on two major discoveries: (1) unique developmental programs underlying the postnatal expression of ion channels responsible for  $\text{Na}$  absorption (ENaC) and  $\text{K}$  secretion (SK/ROMK and BK channels) in this epithelium, thus establishing the physiological basis for total body  $\text{Na}$  and  $\text{K}$  retention required for somatic growth and maintenance of blood pressure, and (2) the role of variations in urinary flow rate (i.e., hydrodynamic forces) in mechanoregulation of renal epithelial ion channels in health and disease. Recent efforts have been devoted to developing model systems, including 3D bioprinted collecting ducts and human PSC-derived kidney organoids, which recapitulate ion transport and signaling phenotypes of the in vivo distal nephron. As the lab serves as a national "single nephron physiology" Core of an NIH-funded O'Brien Center for Kidney

Research, the techniques available to lab members and external investigators include in vitro microperfusion of single nephron segments, fluorescent functional imaging of single cells in native tissue (for measurement of cell pH, calcium, K<sup>+</sup> and Na<sup>+</sup>), patch clamp studies of single cells for analysis of channel activity, and molecular techniques (real time PCR, immunoblotting) applied to single cells and tubules.

**John Cijiang He, MD, PhD**

Professor and Chief, Division of Nephrology  
Department of Medicine (Nephrology)  
Department of Pharmacological Sciences

**Research Interest:** Research in the Cijiang laboratory focuses on podocyte biology and pathology and use of systems biology approach to study signaling networks in kidney disease. Studies in their lab have shown that retinoid acid can reverse abnormal phenotype in diseased podocytes. Retinoic acid, through binding to RARalpha, stimulates intracellular cAMP production and activates CREB and KLF15 transcription factors, leading to podocyte differentiation. They are further studying the role of RARalpha using transgenic and knockout approaches and are also investigating the role of KLF15 in podocyte differentiation. In other studies, they found that AGE causes podocyte apoptosis via activation FOXO4 transcription factor. The activity of FOXO4 is regulated by Sirt-1 through deacetylation. They are now investigating the role of Sirt1-FOXO4 in podocyte apoptosis in diabetic nephropathy. Using systems biology approach, they identified HIPK2 as a key signaling molecule mediating TGF-beta signaling and kidney fibrosis and are currently investigating the role of HIPK2 in epithelial-mesenchymal transformation and renal fibrosis. The Cijiang laboratory is also trying to establish an in vitro model of glomerular filtration unit using nanotechnology, computational modeling, and high-resolution in vivo image studies. This approach will be used to better understand the relationships between the morphology of podocyte foot process, cell signaling, and function, and study the interaction between podocytes, GBM, and endothelial cells.

**Girish N Nadkarni, MD, MPH, CPH**

Professor and System Chair  
Department of Artificial Intelligence and Human Health  
Department of Medicine (Nephrology)

**Research Interest:** Research in the Nadkarni laboratory focuses on using big data and machine learning, especially in the field of genomics and electronic health record data, to improve human health. A recent area of focus is also the utilization of multi-modal data including sensors, wearables and mobile applications for patient engagement and risk prediction. Dr. Nadkarni is core faculty in the Charles Bronfman Institute of Personalized Medicine, which is responsible for the institutional biobank, a cohort of >50,000 diverse ancestry participants with linked genomic and electronic health record data. He is also responsible for the Mount Sinai Digital Health Cohort which is an inception cohort of Mount Sinai patients who are generating unprecedented patient-generated data through wearables, mobile applications and home sensors.

**Evren Azeloglu, PhD**

Professor  
Department of Medicine (Nephrology)  
Department of Artificial Intelligence and Human Health  
Department of Pharmacological Sciences

**Research Interest:** Research in the Systems Bioengineering Laboratory aims at understanding cellular decision-making capabilities related to biomechanical and electrochemical signal processing. They employ multiscale experimental and computational techniques as well as microfabrication and tissue engineering methods to study cell signaling, biomechanics, tissue function and regeneration using cardiovascular and renal model systems. In these studies, they also use multi-platform omics technologies and network analyses for identification of key information processing hubs and regulatory motifs that can be used as therapeutic targets in drug development for complex diseases such as hypertension, diabetic nephropathy and heart failure.

**Paolo Cravedi, MD, PhD**

Associate Professor

Department of Medicine (Nephrology)

**Research Interest:** The Cravedi laboratory is interested in translational research in transplantation and immune-mediated glomerular diseases. The two main ongoing projects focus on the immune effects of erythropoietin and complement and how targeting the related pathways improve outcomes of transplanted organs and glomerulopathies. Through collaborative studies with US and European centers they are also studying biomarkers as risk assessment tools for allograft injury in kidney transplant patients. Before joining Mount Sinai, Dr. Cravedi worked with Dr. Remuzzi in Bergamo, Italy, on clinical studies to minimize immunosuppression in kidney transplantation and he has been involved in the early studies showing the efficacy of B cell depleting therapies in patients with membranous nephropathy and FSGS.

**Ilse Daehn, PhD**

Associate Professor

Department of Medicine (Nephrology)

**Research Interest:** Research in the Daehn laboratory explores the complexity of signaling crosstalk between glomerular cells in the kidney disease. By examining the molecular mechanisms in the glomerulus that result in progression kidney disease, with particular focus on the impact of mitochondrial function, Dr. Daehn hopes to identify novel therapeutic targets and biomarkers.

**Miguel Fribourg, PhD**

Associate Professor

Department of Medicine

**Research Interest:** Research in the Fribourg laboratory focuses on applying quantitative methods to study cellular signaling in the immune system, understand immune disease, and guide clinical intervention. Multidisciplinary approaches that combine theoretical and experimental techniques are critical to study fundamental immune signaling questions in organ transplantation immunology. The Fribourg lab focuses on applying computational modeling, functional genomics, cell signaling, and transplant immunology to identify the molecular mechanisms by which exogenous interferon beta (IFN), a type I interferon, acts on T cells to prolong allograft survival.

**Gabriele L. Gusella, PhD**

Associate Professor

Department of Medicine (Nephrology)

**Research Interest:** The Gusella laboratory is interested in the pathogenetic mechanisms underlying Autosomal Dominant Polycystic Kidney Disease (ADPKD), which is caused by mutations on the PKD1 or PKD2 genes, which code for PC1 and PC2 respectively. In particular, they are focusing on the early events of cystogenesis and the molecular and cellular responses immediately dependent on PKD1 dysregulation. Alteration of the extracellular matrix (ECM) has long been recognized as a distinctive feature of ADPKD epithelia, and the cystogenic process is associated with increased expression of integrins. They recently showed that integrin- $\beta$ 1 is a previously unrecognized key mediator of cystic pathogenesis. Their working hypothesis is that ECM-Integrin-PC1 interactions are important in the cystic pathways of both ciliary and non-ciliary origin. This hypothesis will be tested both in vitro and in vivo. One of the main projects of the Gusella lab aims to elucidate how integrin- $\beta$ 1 functions are affected by PC1 through the comparative analysis of the proteins that participate in the integrin- $\beta$ 1 activation pathway in normal and cystic cells. A second project focuses on the role of integrin- $\beta$ 1 in renal cystogenesis from ciliary defects. Most ciliopathies in which the failure to assemble a normal primary cilium present a cystic kidney phenotype. They study the effects of the deletion of integrin- $\beta$ 1 on Ift88 ciliary mutant using both genetic murine models of renal cystic disease and 3D systems. Results from these studies will provide insights on the function of integrin- $\beta$ 1 in ADPKD pathogenesis and whether cystogenic mechanisms of different genetic origin may converge onto integrin- $\beta$ 1 signaling pathway. Importantly, this work will determine whether the integrin pathway can be pharmacologically targeted to slow the progression of ADPKD.

**Lewis Kaufman, MD**

Professor

Department of Medicine (Nephrology)

**Research Interest:** Dr. Kaufman directs an NIH-funded research laboratory focused on identifying podocyte protective signaling pathways. Their goal is to devise novel therapeutic approaches that pharmacologically augment these protective pathways in proteinuric kidney disease patients, a podocyte targeted approach. To identify such signals, they performed a large-scale functional mutagenic screen to detect genes that when mutated could restore injury susceptibility to genetically injury resistant podocytes. In this way, they discovered several candidate pathways that strongly protect podocytes in the setting of injury. Studies of several of these pathways are ongoing in the Kaufman lab using a variety of methods including cell culture and mouse models, as well as corroborating findings on human kidney material.

**Kyung Lee, PhD**

Associate Professor

Department of Medicine (Nephrology)

**Research Interest:** Research in the Lee laboratory focuses on elucidating the molecular mechanisms driving the progression of chronic kidney disease (CKD), ultimately for the identification of new therapeutic targets to combat the disease progression. The scope of their research encompasses a wide range of molecular pathways driving CKD and kidney fibrosis, which includes diabetic kidney disease (DKD), HIV-associated nephropathy (HIVAN), and focal segmental sclerosis (FSGS). Using an unbiased gene expression analysis of kidney cells from mouse models of DKD, they recently identified secreted molecule LRG1 as a potent angiogenic molecule and inducer of glomerular endothelial cell injury. Their study indicates that its increased expression accompanies accelerated DKD progression in mice and humans. As an important modulator of TGF- $\beta$  signal transduction, LRG1 also contributes to tubulointerstitial fibrosis development in CKD. They are currently working towards the development of LRG1 antagonists to mitigate CKD and fibrosis development.

**Nicholas Chun, MD**

Associate Professor

Department of Medicine (Nephrology)

**Research Interest:** Dr. Chun's current research interests include (a) understanding how cold storage of donor organs prior to transplant alters their interaction with the immune system, and (b) studying how subcellular particles, such as exosomes, mediate priming or control of the immune system.

**Stony Brook University - Renaissance School of Medicine****Sandeep Mallipattu, MD**

Professor

Department of Medicine

Division of Nephrology and Hypertension

**Research Interest:** Research in the Mallipattu laboratory focuses on investigating the molecular mechanisms involved in the development and progression of chronic kidney disease. They have found evidence that a highly specialized family of transcription factors, Krüppel-Like Factors (KLFs), are critically involved in regulating fundamental cellular processes in the kidney: Cell differentiation and proliferation, and cell metabolism. His laboratory is using in vitro and in vivo approaches to establish the physiological and pathophysiological functions of KLFs in various disease processes (Diabetic Kidney Disease, Focal Segmental Glomerulosclerosis, Acute Kidney Injury transition to Kidney Fibrosis) and is utilizing high-throughput screening strategies to identify small molecular compounds that might serve as novel therapeutic agents in disease based on their ability to modulate the expression and function of KLFs. Based on their increasing knowledge of regulators of gene expression, the Mallipattu laboratory is also developing a combinatorial approach to building a platform for kidney regeneration.

**Ryan Williams, PhD**

Associate Professor

Department of Medicine

Division of Nephrology &amp; Hypertension

**Research Interest:** The Williams Nano Lab focuses on the design and translation of nanomedicines to treat and diagnose kidney diseases and related conditions. We invented a kidney-targeting nanoparticle platform which we use to deliver gene therapies and small molecules that can prevent or reverse kidney damage. We have also innovated an implantable fluorescent nanosensor platform to measure levels of inflammatory cytokines and other disease biomarkers. In all of our projects, we aim to develop technologies with the goal of improving patient care.

**Shipra Agrawal, PhD**

Assistant Professor

Department of Medicine

Division of Nephrology &amp; Hypertension

**Research Interest:** The central theme of research in the Agrawal laboratory is to identify and understand the molecular mechanisms and pathways in podocytes and neighboring cells that can be targeted to develop novel therapies for glomerular disease. The additional translational focus is to correlate the basic research findings in patients, and to identify biomarkers for glomerular disease diagnosis and prognosis. Current studies underlying mechanisms and role of selective modulation of nuclear receptor PPAR $\gamma$  and of RNA-mediated mechanisms, such as mRNA alternative splicing and polyadenylation and microRNA functions in podocyte biology and glomerular disease.

**Sian Piret, PhD**

Assistant Professor

Department of Medicine

Division of Nephrology &amp; Hypertension

**Research Interest:** Research in the Piret laboratory aims at understanding the molecular and cellular changes that occur in kidney proximal tubules in acute kidney injury (AKI) and progression of AKI to chronic kidney disease (CKD). Specifically, in her laboratory they are investigating: 1) the roles of members of the Krüppel-like factor (KLF) family of transcription factors, in controlling cellular metabolism in normal kidney, and during AKI and CKD; 2) the role of branched-chain amino acid catabolism in severity of, and recovery after, AKI; and 3) developing novel in vitro and cellular kinase assays for future screening of small molecule inhibitors of branched-chain ketoacid dehydrogenase kinase (BCKDK).

**Karam Aboudehen, PhD**

Assistant Professor

Department of Medicine

Division of Nephrology and Hypertension

**Research Interest:** Research in our laboratory focuses on investigating long noncoding RNAs (lncRNA) in polycystic kidney disease (PKD). We aim to discover therapeutically targetable lncRNAs that prevent or mitigate cyst formation and/or progression in PKD. We are the first group to study the impact of lncRNAs in PKD. We discovered several lncRNAs that play a role in the progression and /or development of PKD. This is a high-impact area, in part because of the combined novelty and mysteries of the roles of lncRNA in PKD. We employ in vitro and in vivo mechanistic studies to decipher lncRNA biology and its impact in PKD.

**Siu Chiu Chan, PhD**

Assistant Professor

Department of Medicine

Division of Nephrology and Hypertension



**Research Interests:** The Chan Lab investigates how dysregulated transcriptional programs drive the initiation and progression of polycystic kidney disease (PKD). Our work aims to uncover the transcriptional and epigenetic mechanisms that govern cyst initiation, early lineage changes, and the transition to progressive cyst expansion. We combine genome engineering, molecular and cellular biology, and biochemical methods with state-of-the-art next-generation sequencing and mass spectrometry-based proteomics to dissect the regulatory networks altered in PKD. By defining these pathogenic regulatory pathways, our long-term goal is to develop targeted therapeutic strategies to prevent cyst initiation and/or halt disease progression in PKD.