RENAL-ELECTROLYTE RESEARCH
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Potassium and Its Role in Hypertension and Chronic Kidney Disease

In the mass media and the layperson's press, a high sodium diet garners the most attention with respect to its contribution to hypertension, heart disease, and kidney health. The media forgets to mention the critical importance of dietary potassium. Research has shown that increasing dietary potassium, despite high sodium intake, is sufficient to reduce blood pressure and can protect against heart disease and chronic kidney disease.

For **Cary Boyd-Shiwarski, MD, PhD**, an assistant professor of medicine in the Renal-Electrolyte Division, it is all about potassium and its role in kidney health and function. Dr. Boyd-Shiwarski joined the Division as a faculty member in 2016 after completing her renal-electrolyte fellowship in the Division, in addition to completing both her medical degree and residency at the University of Pittsburgh School of Medicine.

What drives the majority of Dr. Boyd-Shiwarski's research is the fundamental importance of one's diet to blood pressure and renal health and function. Clinically, Dr. Boyd-Shiwarski is most interested in the management of hypertension and chronic kidney disease (CKD), and she also has an active practice caring for patients with inherited conditions that can drive electrolyte imbalances and CKD, such as Fabry disease, Gitelman syndrome, and Alport syndrome.

Dr. Boyd-Shiwarski's research interests generally revolve around potassium homeostasis and ion transport, with a focus on the regulation of the thiazide-sensitive sodium-chloride cotransporter, NCC, by WNK (With-No-Lysine) kinases. Activating mutations to WNK kinases are known to cause hypertension and hyperkalemia in humans, through activation of NCC. These patients are exquisitely sensitive to treatment with thiazide diuretics. Some of Dr. Boyd-Shiwarski's recent studies have focused on the role of a kidney-specific isoform of WNK1 to facilitate the formation of a protein complex in response to a low potassium diet.

In 2018, Dr. Boyd-Shiwarski secured K08 funding from the National Institutes of Health to support her continuing research into a phenomenon specific to kidney function and potassium homeostasis known as WNK body formation (discussion on Page 6).

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Acute Kidney Injury: A Multispecialty Clinical Problem

Acute Kidney Injury (AKI) in all its forms and with all of its causes is a veritable plague among certain populations of patients, such as the critically ill. There are no effective treatments for it once it occurs. Morbidity and mortality are significant components. A prior AKI leaves one susceptible to re-injury and chronic kidney disease, among other complications.

AKI also is bigger than for any one specialty or subspecialty. It is a problem, if it is to be solved, for every group of clinicians that it touches: nephrology, critical care medicine, cardiology, surgery, pharmacy, radiology, heath information technology, and others. The complexity and challenges presented by AKI — the forms it takes, the causes that drive it, the mechanisms that may mitigate or stop its progression — will only be solved by a coordinated and collaborative effort between disciplines.

UPMC has been at the forefront of AKI research and treatment for decades, and numerous advances have occurred in recent years that are helping to drive forward our ability to prevent, diagnose, and treat the condition.

John A. Kellum, MD, professor of critical care medicine and medicine, vice chair of the Department of Critical Care Medicine, and director of the Center for Critical Care Nephrology, is a leader in AKI research and clinical care at UPMC and internationally. While known for his work in AKI, Dr. Kellum's research and clinical practice span various aspects of critical care medicine, with a focus on sepsis and acute organ dysfunction. At UPMC, he has organized multidisciplinary teams of investigators to study novel approaches to the treatment of sepsis and the understanding of the pathogenesis of AKI. His laboratory integrates the work of epidemiology and health service research with studies of basic mechanisms of disease and new methods of treatment.

Dr. Kellum has been involved in much of the AKI research and clinical program development over many years but is quick to emphasize that the problem is larger than one department and one group of patients, and the global UPMC response to the problem has been an ongoing collaborative effort over many years.

Recent advances in early detection, diagnosis, and clinical tools development are discussed below in this first part of a two-part article. Part two, to be published in Fall 2019, will discuss clinical and translational studies that have shown promise in tackling various aspects of AKI at UPMC and the University of Pittsburgh.

Implementing a Clinical Decision Support System

The successes and advances in AKI research and treatment at UPMC and the University of Pittsburgh are due in no small part to the way leadership in the Renal-Electrolyte Division and Department of Critical Care Medicine have come together to solve a complex problem such as AKI.

"AKI is a problem that requires a coordinated effort across multiple specialties, and I think we have been very successful in making that happen. It shows up in the aspects of the problem that we have tackled, and it shows up in the successes we have had," says Dr. Kellum.

In 2018, Dr. Kellum and colleagues published findings in the *Journal of the American Society of Nephrology* analyzing data for three years on the effect of a clinical decision support system (CDSS) in use at UPMC for AKI.[1]

The computer-assisted decision support tool was designed to derive a baseline serum creatinine level for patients from historical values in the electronic medical record, and then flag changes in the patient's creatinine and KDIGO stage.

While kidney function is monitored using simple blood tests, subtle changes can elude or delay detection of a problem. Failure to recognize and manage acute kidney injury in the early stages can lead to devastating outcomes for patients and increased costs to the health care system. Benefits of earlier detection of AKI include earlier intervention to mitigate loss of kidney function, and reduced hospital and long-term health care costs as a result of avoiding progression to severe and permanent kidney damage.

In 2013, working with UPMC's eRecord system, Kellum's team released a computer program within the electronic health record system across 14 UPMC hospitals. The program monitored levels of blood creatinine, a standard measure of kidney function, over time and analyzed changes in those levels. If the levels rose too high or fast, the program fired an alert in the patient's electronic health record informing doctors that acute kidney injury could be present. It also helped determine the stage of injury based on changes from the patient's baseline kidney function.

To determine what effect the computer program had on physician behavior and patient outcomes, Dr. Kellum and his colleagues analyzed records from more than half a million patients admitted to UPMC. They started a year before the alert system was deployed, and continued for two years after. Patients with acute kidney injury had a small yet sustained decrease in hospital mortality of 0.8 percent, 0.3-day shorter length of stay, and a decrease of 2.7 percent in dialysis rates, compared to patients with AKI prior to alert implementation. Even after adjusting for age and severity of illness, these changes remained highly significant.

What the analysis showed was a small, yet important benefit to the use of such a decision tool. In absolute terms, the changes are small, but given the annual frequency of acute kidney injury in hospitalized U.S. patients of about 12 percent — or 2.2 million people — these results would, if generalized to the entire country, translate into more than 17,000 lives and $1.2 billion saved per year.

"We were able to show a number of outcomes with the study, but also continue to paint the picture of AKI as a prevalent disease process, but one in which one small solution to a large problem can chip away at it and make incremental gains. Ultimately that may be the best strategy to effective AKI prevention, detection, and treatment — many small successes each tackling a subset of the larger problem," says Dr. Kellum.

Efforts in Diagnosis

In 2014, the FDA approved NephroCheck,® the first-ever FDA-approved diagnostic test for acute kidney injury. Dr. Kellum was the lead investigator on the project, and UPMC and the University of Pittsburgh led efforts to discover and validate the biomarkers used in this novel diagnostic tool.

"This was a hugely important step in the battle against AKI. We have been using the test clinically at UPMC since its approval, and we have incorporated it into a variety of therapeutic bundles, particularly in the intensive care unit."

The test analyzes two distinct biomarkers of AKI — TIMP2 and IGFBP7 — and is used to assess which patients are at risk for developing AKI while in the hospital.

Collaborations With Clinical Pharmacology

The most common cause of AKI is nephrotoxic medications. Many patients admitted to the hospital or intensive care unit receive multiple nephrotoxic medications, putting them at significant risk for AKI.

Emily Joyce, MD, a pediatric nephrologist at UPMC Children's Hospital of Pittsburgh, has collaborated extensively with Dr. Kellum on various research projects in AKI. Together they have developed a high-density intensive care patient database of critically ill children that includes data points on more than 12,000 patient encounters over a five-year period, from 2010 to 2014. Dr. Joyce is using the database to understand the associations between the administration of certain medications and AKI to better understand risk stratification, and how the risk of AKI can be minimized in certain medication scenarios. Dr. Joyce's initial investigations are probing antibiotics and antibiotic combinations associated with AKI in critical illness, particularly the use of the broad-spectrum antibiotic vancomycin — alone, and in combination with other agents including piperacillin and tazobactam.

"We also are looking at this in adults with a number of ongoing collaborations Sandra L. Kane-Gill, PharmD, MS, FCCM, FCCP, from the University of Pittsburgh School of Pharmacy. Clearly, it is a much bigger problem than I think anybody recognized. Efforts to mitigate the impact of these medication-induced AKI events also are underway," says Dr. Kellum.

BEACON and PRESERVE Trials

The Biomarker Effectiveness Analysis in Contrast Nephropathy (BEACON) study for which Dr. Kellum is a co-investigator is examining contrast-induced acute kidney injury (CIAKI), which is a serious complication occurring in patients with chronic kidney disease who undergo angiography. The ongoing study led by Dr. Raghavan Murugan, in the Department of Critical Care Medicine is a collaboration with the leaders of the PRESERVE trial, Drs. Steve Weisbord and Paul Palevsky from the Renal-Electrolyte Division. The study seeks to address two primary issues. First, early detection of CIAKI after contrast exposure is problematic because a rise in serum creatinine or a decline in urine output occurs over several days, leading to many missed cases. Additionally, early risk stratification for long-term adverse events poses a challenge because existing risk prediction models are only partially viable. Having a biomarker or markers that detect subclinical CIAKI before creatinine levels and also aid in risk stratification will change the primary and secondary prevention strategies for CIAKI.

"The BEACON trial is partly related to understanding for whom the nephrotoxicity from contrast could be anticipated because of their biomarker signature. As a consequence, we are looking at a variety of markers of kidney injury in this investigation," says Dr. Kellum.

Clinical Guidelines Set for Update

In 2012, Dr. Kellum was co-chair of the Kidney Disease: Improving Global Outcomes (KDIGO) clinical practice guidelines for AKI. In 2019, Dr. Kellum once again will contribute to a new initiative that will update the guidelines.

"Much of the initiatives and work discussed above, and others, will become codified in the new guidelines. In this sense, some of the pioneering work that we have accomplished in AKI at UPMC will become part of the standard therapeutic measures recommended for clinicians globally," says Dr. Kellum.

For example, he and colleagues across the system, most notably Michael Moritz, MD, clinical director of pediatric nephrology at UPMC Children's, have been involved in a two-decades-long effort to define optimal fluid therapies for hospitalized patients. Dr. Moritz's work on fluid tonicity has led to, among other things, the inclusion of the recommendations for isotonic fluids in the recently released American Academy of Pediatrics (AAP) first-ever clinical practice guidelines[2] in the United States for the use of intravenous maintenance fluids in children. The new, evidence-based guidelines are meant, in part, to reduce or prevent as many cases as possible of hyponatremia and its frequently severe morbidities and mortalities. Similarly, Dr. Kellum's work on chloride concentration in fluids has led to recent large pragmatic studies that have concluded that saline should be avoided in favor of more physiologic isotonic crystalloids.

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Probing the Molecular World of Acute Kidney Injury and Chronic Kidney Disease

Acute kidney injury (AKI) continues to be a clinical challenge. At present, AKI has no effective treatments, and severe or repeated episodes of AKI can lead to chronic kidney disease (CKD) and irreversible kidney scarring. In fact, individuals with a history of AKI are at a higher risk of sustaining a second or third injury, compounding the short- and long-term consequences.

CKD also is limited in its treatment — most individuals are treated with angiotensin converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARB), but these are only partially effective and come with significant side effects. Much research and progress is underway to better understand the cellular and molecular mechanisms driving AKI/CKD, their various phenotypes, targets for therapeutic intervention, and early detection modalities.

Roderick Tan, MD, PhD, is a physician-scientist who is working to unravel the molecular mechanisms underlying the development of AKI, CKD, and fibrosis utilizing both in vivo and in vitro approaches. Dr. Tan is assessing novel ways in which the glomerular and tubular compartment cross-talk in disease, and how the Keap1/Nrf2 pathway is involved in AKI/CKD. Dr. Tan has also studied the Wnt/ß-catenin pathway and how matrix metalloproteinases affect renal injury. More recently, Dr. Tan has begun to study the renal microvasculature using novel ultrasound imaging technology in collaboration with Kang Kim, PhD, from the University of Pittsburgh.

Keap1 and the Nrf2 Pathway in AKI and CKD

The Keap1 and Nrf2 proteins work in tandem in a pathway that modulates oxidative stress, inflammation, and cytoprotective mechanisms in the kidney. Dr. Tan's research has analyzed the roles of Keap1 and Nrf2 in murine models of ischemia-reperfusion injury (IRI) and unilateral ureteral obstruction (UUO), and how this leads to fibrosis and ultimately CKD. Nrf2, or nuclear factor erythroid 2, is a transcription factor that enhances antioxidant responses and is also associated with an anti-inflammatory response via effects on NFκB. The Keap1 (Kelch-like ECH associated protein-1) protein functions as an inhibitor of Nrf2 function.

Dr. Tan utilizes Keap1 hypomorph murine models from the lab of Thomas W. Kensler, PhD, formerly with the University of Pittsburgh Department of Pharmacology and Chemical Biology, but now at the University of Washington, who also studies the Keap1/Nrf2 pathway. These models have been genetically engineered to underproduce or under-express the Keap1 protein.

A decrease in expression of Keap1 leads to increased Nrf2 activity. In models of IRI and UUO, Dr. Tan and his study team have shown that Nrf2 hyperactivity leads to a protective effect in the kidney that can mitigate the injury and stop the progression to fibrosis and CKD.

"By hyperactivating the Nrf2 transcription factor through reduction of Keap1 levels, we were able to show that after IRI there is protection against the development of scarring or fibrosis and CKD. This may prove to be a viable therapeutic target in the future — activating the Nrf2 pathway in the setting of IRI," says Dr. Tan.

His studies also investigated this protective effect in the setting of UUO. While the mechanism of injury is different, the protective effect was confirmed as fibrosis and progression to CKD were inhibited by hyperactivation of Nrf2.

Keap1, Nrf2, and Proteinuria

Dr. Tan's research adds to a large body of literature suggesting that pharmacologic activation of the Keap1/Nrf2 pathway can be protective in AKI. However, there are many different pathways leading to kidney injury or damage. One such pathway is glomerular disease leading to proteinuria.

In early clinical trials in patients with diabetic kidney disease and proteinuria, the enhancement of the Nrf2 pathway showed increases in kidney filtration function, but the studies also showed an increase in proteinuria, which would be expected to worsen disease in the long run.

Dr. Tan's research group is interested in this conundrum.

"We exposed our Keap1 hypomorphs to a number of proteinuric injuries. Instead of being protected, these mice actually exhibited worse proteinuria. So, while Nrf2 activity appears to be protective in AKI, it appears that for proteinuric kidney disease the opposite is true. We have presented our research at an international kidney conference and have submitted our manuscript for publication," says Dr. Tan.

Studying the Microvasculature of the Kidney

In 2018, Dr. Tan was awarded the prestigious Edith H. Blattner Young Investigator Grant from the National Kidney Foundation (NKF), one of several grants awarded every year through the NKF Young Investigator Research Grant Program. Dr. Tan's award will support new research he is conducting that will examine the microvasculature of the kidney before and after injury using high-resolution ultrasound technology coupled with the use of a microbubble contrast agent. Dr. Tan's collaborator on the project is **Kang Kim, PhD**, associate professor of medicine and bioengineering at the University of Pittsburgh and the UPMC Heart and Vascular Institute. Dr. Kang is an expert in the use of this high-resolution ultrasound technology and microbubble contrast agent, and he is involved in numerous studies across the University and UPMC that is employing this imaging modality to gain new insights.

Past studies in animal models have shown that the density of the renal microvasculature (the small blood vessels of the kidney) becomes decreased after an AKI caused by IRI. This decrease in the vascularity of the

kidney leads to a decrease in perfusion and oxygenation, making the organ more prone to future IRI or the development of CKD.

Studying this in humans has always been difficult and confined mainly to postmortem biopsy and analysis. However, that may be changing with the advent of high-resolution ultrasound combined with microbubble contrast agent. This technique is routinely used clinically in the field of cardiology.

"We currently are working on these studies in animal models with Dr. Kim, and that work is funded through a pilot award from the Pittsburgh Center for Kidney Research P30 grant. Our new NKF grant will allow us to pilot a study in human subjects whom we hope to soon begin enrolling," says Dr. Tan.

Dr. Tan's goal is to eventually study AKI patients in the hospital, examine their renal microvasculature, and use the findings as a prognostic tool to determine who will likely recover their kidney function and who will progress to CKD.

"As clinicians we see the spectrum of AKI and have a sense for which patients may end up on a particular trajectory, but we do not have good objective measures to accurately tell us who will get better or worse. I am hopeful that this research will eventually lead to a more quantitative measure of an AKI patient's kidney prognosis," says Dr. Tan.

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UPMC Physician Resources

For the latest CME courses, videos, news, and events from the Renal-Electrolyte Division, visit **UPMCPhysicianResources.com/Kidney**.

Current CME Courses Include:

Cancer and Renal Transplantation

Presented by Sundaram Hariharan, MD

Dr. Hariharan discusses the importance of cancer after kidney transplantation and how it impedes long-term kidney transplant survival.

Calcium and the Elderly

Presented by James R. Johnston, MD

Dr. Johnston gives a presentation on normal calcium metabolism and an approach to calcium in the elderly.

Renal Year in Review

Presented by Manisha Jhamb, MD, MPH

Dr. Jhamb discusses four research studies and their implications, including preventive management of IV contrast-associated AKI; managing depression in patients with chronic kidney disease; the effect of intensive hypertension management on long-term kidney function; renal effects of sodium glucose cotransporter 2 (SGLT2) inhibitors.

Immunosuppression in Renal Transplantation — Successes and Challenges

Presented by Rajil Mehta, MD, FASN

Dr. Mehta outlines the mechanisms for T cell immunosuppression, induction therapy, and situations for use of mTOR inhibitors in kidney transplantation.

Video Rounds

Video Rounds is a series of short, informative, and educational videos created for physicians and covering a variety of medical and surgical disciplines. New topics from the Renal-Electrolyte Division include:

Hypernatremia Treatment

Presented by Helbert Rondon-Berrios, MD

Renal Palliative Care

Presented by Jane Schell, MD

Potassium and Its Role in Hypertension and Chronic Kidney Disease *Continued from Page 1*

The Importance of Potassium on Kidney Function and Health

For more than 100 years the importance of dietary potassium has been recognized for its ability to act as a diuretic and reduce blood pressure. Still, according to recent research, a full 98 percent of the United States population does not consume the recommended daily intake of this important element.

"Because I'm interested at a fundamental level on what will keep the kidneys healthy, and how they function at the cellular and molecular levels, my research uses mouse models to study the effects of various types of potassium (e.g., potassium depletion, potassium citrate, potassium chloride) and their effect on hypertension, kidney function, and health," says Dr. Boyd-Shiwarski.

Her animal model experiments assess blood pressure, weight change, and serum levels of sodium and potassium with the long-term goal of determining how various diets and levels of potassium affect kidney function.

The health of the kidney in relation to potassium intake also plays a role in Dr. Boyd-Shiwarski's research. Her research assesses WNK kinases that regulate sodium transport in the kidney and how they are affected by varying levels of potassium intake relative to various overall dietary loading schemes to develop a better understanding of how a normal kidney responds to different potassium diets.

"My new K08 grant is designed — at a general level — to investigate how the kidney senses potassium levels and then translate this information into blood pressure and kidney health. We think WNK kinases are one of the mechanisms by which the kidney senses potassium. These kinases sense changes in potassium levels and then coordinate volume and potassium homeostasis by regulating renal sodium transport," says Dr. Boyd-Shiwarski.

Her studies on this front involve knockout models that have a deletion for the KS-WNK1 kinase. "With manipulations to dietary potassium, and by varying the potassium anion with either citrate or chloride, we are determining how KS-WNK1 senses potassium and relays that message to downstream sodium transporters. Do the KS-WNK1 knock-out mice sense potassium differently? Is their blood pressure different in response to potassium? Do their kidneys show telltale signs of chronic kidney disease, or are they more resistant? This involves knowing what role these kinases play within the kidney," says Dr. Boyd-Shiwarski.

Dr. Boyd-Shiwarski's research also entails studies to understand how the KS-WNK1 kinase functions in the distal convoluted tubule of the nephron, and how changes in that part of the nephron can affect the entire nephron — in the proximal tubule, the cortical collecting duct, and the loop of Henle.

WNK Bodies and New K08 Research

Creatures — mice, humans — that exist for long periods on a low potassium diet have been found to form what is known as "WNK bodies" in the kidney. These WNK bodies are transient aggregates that form exclusively in the distal convoluted tubules.

"We do not believe these bodies are pathological but are rather a kind of protective phenomenon that occurs when potassium levels are low for extended periods. They can form rapidly when potassium levels decrease substantially, but they also dissipate once normal levels are achieved," says Dr. Boyd-Shiwarski.

Researchers have known about the existence of these WNK bodies for over eight years, and that low levels of potassium in the diet lead to their formation, but until very recently, it was not known what molecular processes drove the formation. That is until Dr. Boyd-Shiwarski and colleagues published on the matter in 2017.[1] What Dr. Boyd-Shiwarski's team found is that the KS-WNK1 kinase is responsible for their formation. The next question for Dr. Boyd-Shiwarski to answer is: Why do they form?

"Why in a low potassium diet do these puncta form in the kidney? This also is a central part of the K08 grant. The first part of the K08 is designed to investigate further how potassium and potassium imbalance regulate blood pressure. The second aspect is understanding why WNK bodies form when potassium is low, and how they affect blood pressure. Our working hypothesis is that they regulate the sodium chloride cotransporter NCC, but we do not understand how … we are trying to assemble the full picture of this process whereby low dietary potassium occurs (i.e., the Western diet) and WNK bodies form that are KS-WNK1-dependent. Again, we believe they serve some form of protective role in the kidney to sense the potassium and then act downstream on the NCC cotransporter to regulate sodium reabsorption, which eventually affects blood pressure. I am excited about this research project because it is possible that we could reduce the burden of hypertension and chronic kidney disease by simply increasing dietary consumption of potassium. Given the significant burden of the two diseases, further research into effective measures to prevent hypertension and chronic kidney disease is needed," says Dr. Boyd-Shiwarski.

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[2] The Function of Kidney-Specific (KS)-WNK1 Condensates During Potassium Stress. NIH Project Number: 1K08DK118211-01.

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Recent Publications from Division Faculty

Researchers in the Renal-Electrolyte Division are engaged in a diverse array of basic science, translational, and clinical investigations on numerous aspects of kidney development and disease. Below is a selection of recent high-impact publications from faculty members.

Lakkis FG, **Chalasani G**, **Hariharan S**. Antibody-Mediated Rejection of Solid-Organ Allografts. *N Engl J Med.* 2018 Dec 27; 379(26): 2580.

Rondon-Berrios H, **Tandukar S**, Mor MK, **Ray EC, Bender FH, Kleyman TR, Weisbord SD**. Urea for the Treatment of Hyponatremia. *Clin J Am Soc Nephrol.* 2018 Nov 7; 13(11): 1627-1632.

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Division News and Notes

Jane Schell, MD, was awarded the Hastings Center Cunniff-Dixon Foundation Early Career Award for her dedication to improving the care of seriously ill patients with kidney disease. The prestigious national award is given annually to five palliative care doctors whose work enriches the relationship of doctors and their patients who are near the end of life. Dr. Schell developed NephroTalk, a nationally recognized curriculum that prepares nephrology fellows to have difficult conversations with patients and families, including end-of-life discussions. She is an associate professor in the Divisions of Renal-Electrolyte and General Internal Medicine.

Notable New Grants

Manisha Jhamb, MD, MPH, and Khaled Abdel-Kader, MD, MS (Vanderbilt Center for Kidney Disease)
Population Health Management to Optimize Care for Patients with High Risk Chronic Kidney Disease.
NIDDK R01 DK116957

Manisha Jhamb, MD, MPH
OPTIMIZing carE in Chronic Kidney Disease (OPTIMIZE CKD).
NIDDK R18 DK118460

Lori Birder, PhD, and **Gerard Apodaca, PhD**
Bladder Mucosal Dysfunction During Aging.
NIA R01 AG056944

Thomas Kleyman, MD
Pittsburgh Center for Kidney Research.
NIDDK P30 DK079307

Thomas Kleyman, MD
Renal and Epithelial Biology Training Program.
NIDDK T32 DK061296

Cary Boyd-Shiwarski, MD, PhD
The Function of Kidney Specific (KS)-WNK1 Condensates During Potassium Stress.
NIDDK K08 DK118211

Roderick Tan, MD, PhD
Tubular to Glomerular Crosstalk in Proteinuric Chronic Kidney Disease.
American Society of Nephrology (ASN) Carl W. Gottschalk Research Scholar Grant

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LIFE CHANGING MEDICINE
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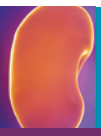

ABOUT THE UPMC RENAL-ELECTROLYTE DIVISION

The Renal-Electrolyte Division is devoted to clinical care and academic excellence, and training the next generation of nephrologists. Our multidisciplinary approach provides the highest quality care for patients with complex kidney and electrolyte disorders.

- Inpatient services at UPMC Presbyterian focus on patients with acute kidney injury, chronic kidney disease, end stage kidney disease, and patients awaiting or who have received kidney transplants. Our on-site dialysis center performs nearly 10,000 dialysis treatments a year in various settings.
- Outpatient services are provided at our specialized kidney and multidisciplinary clinics treating a variety of kidney and hypertensive disorders.
- The Pittsburgh Center for Kidney Research, one of eight nationwide NIDDK-supported George M. O'Brien Kidney Research Core Centers, supports more than 110 investigators and provides funding for pilot projects.

To learn more about the UPMC Renal-Electrolyte Division, please visit **UPMCPhysicianResources.com/Kidney**.

UPMC PHYSICIAN RESOURCES

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For consults and referrals, please call UPMC's 24-hour physician OnDemand service at **1-866-884-8579.**

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