On October 5, 2018, a special Symposium was held in honor of Dr. Navar’s 30th anniversary as Professor and Chair of the Department of Physiology at Tulane University, School of Medicine. The Symposium was a celebration of Dr. Navar’s achievements, leadership, and mentorship over three decades. The one day event consisted of presentations by former trainees and colleagues of Dr. Navar. Former trainees travelled from other cities to attend and present at this symposium, and many who were unable to attend sent greetings, videos, letters, and messages to him that were also recognized at the symposium.

Dr. Luis Gabriel ‘Gabby’ Navar received his PhD in 1966 from the University of Mississippi under the direction of Dr. Arthur Guyton and continued as a postdoctoral fellow and faculty member until 1974. During that time, he also spent a year at Duke University. He then joined the Nephrology Division and Physiology Department at the University of Alabama at Birmingham where he rose to full professorship. In 1988, he joined Tulane University as Professor and Chair of the Department of Physiology. In 2002, he also became Co-Director of the Hypertension and Renal Center of Excellence.

Dr. Navar’s research has focused on the regulation of kidney vascular resistance, the control of sodium excretion and the intrarenal and intratubular generation of Ang II and angiotensinogen in hypertension. He has over 400 peer-reviewed publications including chapters and reviews. His work has been funded primarily by grants from the NHLBI, NIDDK, NCRR and NIGMS.
Dr. Navar is an active member of many societies including the APS, ASH, ASN, AHA, IASH and SSCI. He has served on various study sections (NIH, VA, AHA) and on Editorial Boards for several journals including American Journal of Physiology and Hypertension. He has held leadership roles serving as Councilor and President of the American Physiological Society (APS) and of the Association of Chairs of Departments of Physiology, as well as chair of the Leadership Committee of the Council for High Blood Pressure Research of the AHA and of the Executive Committee of IASH.

With over 40 years of experience in training medical students, graduate students, postdoctoral fellows and faculty in the Physiological Sciences, Dr. Navar trained and supervised over 100 trainees in areas of renal physiology, renin-angiotensin system and pathophysiology of hypertension with many of them being from underrepresented minorities. His former trainees now hold key positions and full professorships in medical schools, research centers, industry and government. His record of mentoring was recognized by APS with the Bodil Schmidt-Nielsen Distinguished Mentor and Scientist award, by Tulane with the Inaugural Oliver Fund Award for Excellence in Faculty Mentoring, and by the 2018 Mentor of the Year Award from SSCI. His research has been recognized by the Gottschalk lectureship, the Robert W. Berliner Award for Excellence in Renal Physiology from the Renal Section, the Ray G. Daggs Award, and the Walter B. Cannon Award from APS as well as the Dahl and Corcoran lectureships and the Excellence Award for Hypertension Research from the Hypertension Council of the AHA.

Pictures taken at the event are displayed in the following pages. Guest speakers at the event included: Edward W. Inscho, PhD (UAB), John D. Imig, PhD (MCW), Jia L. Zhou, MD, PhD (UMC), Matthew Walker, III, PhD (Vanderbilt), Rudy Ortiz, PhD (UCMerced), Lisa Harrison-Bernard, PhD (LSU), Kenneth D. Mitchell, PhD (Tulane), Dewan SA Majid, MD, PhD (Tulane), Ryosuke Sato, PhD (Tulane), Supaporn Kulthinee, PhD (Tulane), Owen Richfield (Tulane), and Minolfa C. Prieto, MD, PhD (Tulane).
Pictures taken during the L. Gabby Navar Symposium of presentations by some of the former trainees.
The celebration continued till the evening with a formal dinner held at the Cardinal Room in Mazarin Hotel in New Orleans. The following day, on October 6, an informal dinner gathering was held at Dr. Minolfa Prieto’s home.
Dr. Paul Whelton, a clinical professor and the Show Chwan Health System Endowed Chair in Global Public Health at Tulane University School of Public Health and Tropical Medicine, was awarded the 2018 AHA Excellence Award for Hypertension Research. The award is considered one of the most prestigious awards for a researcher in the field of hypertension.

Dr. Whelton, along with Drs. Ham, He and Navar, were the founding members of Tulane Center of Biomedical Research Excellence (COBRE) program. Initially formed in 2002 through the support of an institutional planning grant, the Tulane Hypertension and Renal Center of Excellence (THRCE) was in 2002 awarded the NIH, COBRE award in Hypertension and Renal Biology to support the creation and operation of the center's state-of-the-art research facility that would provide an enriched research and mentoring environment for investigators participating in hypertension and renal research. The COBRE funding has successfully generated highly competitive research studies by junior investigators whose COBRE and center supported research were later awarded federal and national funding that allowed them to continue their research in hypertension and related renal and cardiovascular disorders as independent investigators.

In 2018, Dr. Whelton led the team that redefined high blood pressure, lowering the threshold for high blood pressure diagnosis to 130/80 mm Hg instead of 140/90 mm Hg. The new clinical guideline eliminates “prehypertension” recommends earlier and more intensive treatment with lifestyle changes and, in some cases, medication. The goal is to help patients better understand their cardiovascular risk and attain earlier and more effective control of high blood pressure, said Dr. Whelton, chairman of the American Heart Association (AHA)/American College of Cardiology (ACC) Hypertension Guidelines Committee.

The AHA award honors excellence in research and discoveries in the field of hypertension as well as a researcher's contributions. The selection committee assesses the candidates' impact on their fields throughout their productive careers as well as any single discovery.
March 14, 2019 is World Kidney Day! A joint initiative of the International Society of Nephrology (ISN) and the International Federation of Kidney Foundations (IFKF), World Kidney Day (WKD) began in 2006 as a global health awareness campaign that focuses on the importance of kidneys and the mechanisms that reduce kidney disease. WKD has been celebrated ever since, every second Thursday of March, in more than 100 countries on 6 continents. Each year, the campaign focuses on a theme. The 2019 campaign will focus on the burden of kidney disease, disparities and access to healthcare with the theme: Kidney Health for Everyone Everywhere. (Details on WKD can be accessed at: www.worldkidneyday.org).

In honor of WKD, THRCE has scheduled two special events on March 14th:

- From 9am until 3pm, THRCE, the Department of Physiology, the Department of Medicine (Section of Nephrology), and the National Kidney Foundation of Louisiana, will conduct a “Tulane WKD Health Screening Fair” in the Lobby of Tulane University Hospital and Clinics. This free program is designed to screen people at risk for CKD and promote CKD awareness among the public. Participants will be screened for blood pressure & the risk for developing kidney disease.

- At 4pm, a Special WKD THRCE Seminar will be presented by the distinguished Cardiologist, Professor, and the Gerald S. Berenson Endowed Chair in Preventive Cardiology at Tulane, Dr. Keith C. Ferdinand. Dr. Ferdinand is past Chair of the National Forum for Heart Disease and Stroke Prevention and has served as Chief Science Officer and past chair of the Association of Black Cardiologists. He has also served as a board member of the American Society of Hypertension, the Southwest Lipid Association, and the International Society of Hypertension in Blacks. The Seminar will be held in the Pharmacology Conference room, 4700, at Tulane University, School of Medicine.
THRCE PARTicipates in the 
AHA 2018 Heart Walk

THRCE participated in the 2018 Heart Walk sponsored by the American Heart Association on Saturday, November 17th, 2018. The Heart Walk is an annual event to raise money for the American Heart Association. The funds raised is used for critical research and education on cardiovascular diseases. The Heart Walk was held at LaSalle Park in Metairie, Louisiana and included numerous fun-filled health and wellness activities, free food and entertainment. TSOM team coaches and members helped raise over $2,415 for the AHA fundraising campaign. Overall, the AHA Heart Walk, with the fundraising support from Tulane and other companies in New Orleans, raised over raised over $420,000 net; this fund will be used to accomplish the AHA mission of fighting heart disease and stroke in the Greater New Orleans area! The funds raised for the 2018 New Orleans Heart Walk help to fund advocacy programs, train and certify people in CPR, fund lifesaving research, advance wellness in the workplace and in schools, and educate the public on the prevention of heart disease and stroke in the New Orleans Community.
GRANTS, HONORS & RECOGNITION AWARDED TO THRCE AFFILIATED INVESTIGATORS

L. Gabriel Navar, PhD:
- Visited the Medical College of Wisconsin on September 25-26 and presented “Regulation of the intrarenal renin-angiotensin system in hypertension and Diabetes” to the Cardiovascular Center directed by Dr. Ivor J. Benjamin, current AHA president.

Andrei Derbenev, PhD:
- Presented “Novel role of glycine in control of sympathetic outflow,” at Georgia State University in September 2018.
- Article was accepted in the Journal of Physiology London. The title of the article is “Glycinergic neurotransmission in the RVLM controls the time course of baroreflex mediated sympathoinhibition.”

Jean-Pyo Lee, PhD:
- Received a NIH R01 grant award for over $2.4M from the National Institutes of Neurological Disorders and Stroke. The grant is for 5 years and is titled “Combination treatment of ischemic stroke with perlecan DV and neural stem cells.”
- Participated in NIH, Brain Injury and Neurovascular Pathologies (BINP) Study Section, Washington, D.C., October, 2018.

Supaporn Kulthinee, PhD:
- On September 8, presented during the Session, “Renal Hemodynamics and Tubular Transport” at the 2018 AHA's Joint Hypertension Scientific Sessions held in Chicago. The conference is the premier basic and translational Hypertension conference with the AHA's Council on Hypertension, AHA's Council on Kidney in Cardiovascular Disease with over 600 attendees from 22 countries.
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KAILASH N. PANDEY, PHD:
- Appointed to the Editorial Board of Hypertension, November 1, 2018.
- Presented to the Department of Medicinal Chemistry at the School of Ayurvedic Medicine, Institute of Medical Sciences in Banaras Hindu University, Varanasi, India. The talk, “Genetic ablation of Npr 1 provokes immunogenic renal inflammatory responses in hypertensive diseased state,” was presented on December 1, 2018.
- On December 6, presented, “Genetic disruption of Npr1 gene provokes high levels of proinflammatory cytokines and fibrosis in the kidneys of mutant mice,” at the Department of Life Sciences in Amity Science, Technology & Innovation Center at Amity University in Noida-Delhi, India.
- Presented, “Molecular & genetic aspects of atrial natriuretic peptide receptor-A in the regulation of blood pressure and renal function” on December 15th, in the International Symposium on Molecular Medicine at Sanjay Gandhi Postgraduate Institute of Medical Sciences (SGPGIMS) in Lucknow, India.

MINOLFA C. PRIETO, MD, PHD:
- Received a 2018 Research Career Enhancement Award from the American Physiological Society. Dr. Prieto will train with Dr. Ariel Gomez’s group at the University of Virginia.
- Invited to Chair and to present at the the International Society of Hypertension Satellite Symposium on the Renin-Angiotensin-Aldosterone System, that was
Continued...

held in Guangzhuo, China from September 18th-20th and in Beijing from September 20th-25th. The title of her talk was “Impact of plasma soluble prorenin receptor in cardiovascular diseases.”

ZUBAIDA SAIFUDEEN, PHD:
- Presented “Metabolism and nephron progenitor cell fate” as an invited speaker at the University of Helsinki, Finland on October 15th.
- Moderated at the American Society of Nephrology (ASN) meeting in San Diego. The title of the session was “Translational research innovations in kidney organoid and bioengineered model systems.”

ANDREA ZSOMBOK, PHD:
- Received a research supplement from National Institute of Health-NIDDKD for her research titled “TRPV1-Dependent Autonomic Control in Diabetes.” The supplement will provide an additional year of funding for her research project.
- Recognized in the Tulane News in an article titled, “Tulane professor receives NIH supplement for Alzheimer’s research” that was published on November 20th 2018. The news can be accessed at: https://news.tulane.edu/pr/tulane-professor-receives-nih-supplement-alzheimer%E2%80%99s-research.
- Gave an invited seminar on September 10th at Georgia State University. The title of her presentation was, “Brain-liver pathway in diabetes.” Attended an APS Science Policy Committee Meeting Rockville, MD and Capitol Hill Day in Washington DC on September 16th and 17th.
- Participated in an NNRS Study Section in Bethesda, MD on September 26th-28th.
- Attended the Cayman Peptide Conference in Curacao from October 17th-19th.

GRADUATE STUDENTS: OWEN RICHFIELD
- Owen Richfield, (mentor: Dr. Navar), received a Research Fellowship Award from NIH-NIDDK for 2 years for his project titled “Development of a computational biomechanics model of the glomerulus to assess risk of mechanical stress- induced glomerular injury in conditions of reduced afferent arteriole vasoconstrictive response.”
THRCE SPONSORED PRESENTATIONS

Speakers who present a THRCE Seminar-sponsored presentation are asked to provide a brief summary of their talk that we can share with our newsletter audience. From September through December 2018, the following speakers presented THRCE-sponsored seminars:

- **JAMES D. STOCKAND, PH.D.**
  
  *Professor of Cellular & Integrative Physiology*
  *Director of Pathobiology Of Occlusive Vascular Disease Fellowship Program*
  *University of Texas Health Science Center*
  *San Antonio, TX.*

  On Monday, October 15th, 2018, Dr. James Stockand presented a lecture that was sponsored by THRCE and the Departments of Physiology and Medicine titled, “Disruption of Purinergic Control of Sodium Excretion Intrinsic to the Distal Nephron Causes Salt-Sensitive Hypertension.”

**SUMMARY OF PRESENTATION:**

Sodium balance within the human body influences arterial blood pressure where excretion of sodium from the body decreases blood pressure, and concentration of sodium in the body increases blood pressure. Sodium balance, in part, reflects sodium consumption minus sodium excretion. Sodium is primarily excreted from the body by the kidneys. Consequently, inappropriate sodium excretion by the kidneys causes disordered arterial blood pressure. Humans, similar to all other animals that walk on the land, fine tune urinary sodium excretion by modulating the activity of the epithelial Na+ channel (ENaC). The activity of ENaC is under the discretionary control of hormones and factors that match sodium excretion with sodium intake in the maintenance of arterial blood pressure. Consequently, genetic mutations in ENaC and upstream modulators of this channel that cause a gain or
loss of function lead to disordered renal sodium excretion and dependent pathological changes in blood pressure. Liddle’s Syndrome and autosomal recessive pseudohypoaldosteronism type 1 (PHA1) are examples of dominant and recessive forms of familial hypertension and renal salt wasting, respectively, caused by mutation of ENaC. Autosomal dominant PHA1 is a Mendelian form of renal salt wasting caused by loss of function mutation of the receptor for the mineralocorticoid steroid hormone, aldosterone. Aldosterone is the primary systemic hormone that modulates the activity of ENaC. It does so as the final hormone in the renin-AngII-aldosterone system (RAAS). This hormone cascade controls blood pressure through feedback regulation where a fall in arterial blood pressure stimulates renin secretion to increase plasma AngII and aldosterone concentrations with stimulation of ENaC, in part, causing a decrease in sodium excretion in protection of blood pressure. Several contemporary drugs used to treat elevated arterial blood pressure interdict the RAAS. In addition to aldosterone, other systemic hormones and factors modulate sodium balance through the regulation of ENaC. My lab has demonstrated that the activity of ENaC, and consequently, renal sodium excretion and arterial blood pressure, are influenced by the humoral factor, ATP. ATP via cell membrane receptors decreases the activity of ENaC to affect renal sodium excretion. Compelling evidence shows that increases in urinary flow stimulate ATP secretion into the urine. In many instances, urinary flow is a surrogate for arterial blood pressure. As demonstrated in genetically modified mice, loss and stimulation of ATP inhibition of ENaC causes inappropriate sodium retention and wasting, respectively, leading to consequent pathological changes in blood pressure. We find that ATP regulation of ENaC acts in parallel with regulation by aldosterone and is as quantitatively important to the control of arterial blood pressure as this steroid hormone. This research has redefined the paradigm through which we understand the regulation and function of ENaC during the control of blood pressure, identified several novel targets to decrease blood pressure independent of interdicting the RAAS, and provides the causative mechanism underlying some forms of hypertension of previously unrecognized etiology.
Dr. David Stec presented two talks on Monday October 22nd, 2018 that were co-sponsored by THRCE and the Department of Physiology. The talks, “Role of Biliverdin Reductase in Obesity-Induced Renal Injury” was presented at the Renal & Vascular Workshop and “Bilirubin and Biliverdin Reductase in Metabolic Disease” was presented at the noon Physiology Seminar Series meeting.

SUMMARY OF PRESENTATIONS:
“Bilirubin and Biliverdin Reductase in Metabolic Disease”
Obesity is a major public health concern with more than 70 million obese individuals (BMI >30 kg/m2) over 20 years of age in the U.S.. The total number of overweight and obese individuals in the U.S. is estimated to exceed 150 million people. Obesity is the leading cause of non-alcoholic fatty liver disease (NAFLD) which has increased from 46 to 75% of all chronic liver disease from 2005 to 2008. NAFLD also leads to hepatic insulin resistance that contributes to development of type II diabetes, a major risk factor for cardiovascular disease. Thus, development of novel therapies for prevention and reversal of hepatic steatosis and insulin resistance is of critical importance. Several large population studies have shown a strong negative correlation between serum bilirubin levels and protection against development of hepatic steatosis, insulin resistance, and metabolic syndrome. Despite this correlative evidence, there is a knowledge gap in the mechanism responsible for the protection provided by bilirubin and the potential to leverage these mechanisms for treatment of hepatic steatosis. We have data demonstrating that moderate hyperbilirubinemia resulting from direct treatment with bilirubin decreases hepatic steatosis and insulin resistance in mice with established dietary-induced obesity. These data demonstrate that increases in plasma bilirubin levels can be protective against obesity induced hepatic steatosis and insulin resistance. Bilirubin can also be generated via an intracellular mechanism in hepatocytes through the enzyme biliverdin reductase (BVRA). We have developed a novel model of hepatic specific knockout of BVRA, and have exciting data
showing that the liver specific knockout of BVRA exacerbates hepatic steatosis and insulin resistance in response to dietary induced obesity.

There are several potential mechanisms by which bilirubin protects against hepatic steatosis and insulin resistance. Bilirubin is known to be a powerful antioxidant in the body; however, this bile pigment may also have other beneficial actions. We have data indicating that bilirubin is a novel signaling molecule that transactivates the nuclear receptor family of transcription factors. Our data demonstrate that bilirubin activates peroxisome proliferator-activated receptor-a (PPARa) transcription, which induces beta-oxidation of lipids by activation of genes such as CPT1, GS2 and FGF21. These data are the first direct evidence that bilirubin can act as a signaling molecule. We also have preliminary data demonstrating that hepatic bilirubin generation from BVRA also protects PPARa function by attenuating the levels of glycogen synthase kinase-3b (GSK3b) in the liver. We are actively looking for novel ways to increase serum bilirubin levels as well as examining how to alter the levels of BVRA in the liver as a potential treatments for obesity-induced NAFLD.

“Role of Biliverdin Reductase in Obesity-Induced Renal Injury”

Obesity and increased lipid availability have been implicated in the development and progression of chronic kidney disease. One of the major sites of renal lipid accumulation is in the proximal tubule cells of the kidney, suggesting that these cells may be susceptible to lipotoxicity. We previously demonstrated that loss of hepatic BVRA causes fat accumulation in livers of mice on a high-fat diet. To determine the role of BVRA in mouse proximal tubule cells, we generated a CRISPR targeting BVRA for a knock-out in mouse MCT proximal tubule cells (BVRA KO). The BVRA-KO cells had significantly less metabolic potential and mitochondrial respiration, which was exacerbated by treatment with saturated fatty acid, palmitic acid. The BVRA KO cells also showed increased intracellular triglycerides which were associated with higher fatty acid uptake gene cluster of differentiation 36 (CD36) as well as increased de novo lipogenesis as measured by higher neutral lipids. Additionally, NGAL1 expression, Annexin-v FITC staining, and LDH assays all demonstrated that BVRA KO cells are more sensitive to palmitic acid-induced lipotoxicity than wild-type cells. Phosphorylation of BAD, which plays a role in cell survival pathways, was significantly reduced in palmitic acid treated BVRA KO cells. These data demonstrate the protective role of BVRA in proximal tubule cells against saturated fatty acid-induced lipotoxicity and suggest that activating BVRA could provide a therapeutic in protecting from obesity-induced kidney injury. We have also developed novel mouse models of BVRA KO in different nephron...
segments and are currently investigating the role of BVRA in the development of dietary obesity-induced kidney injury.

**JONATHAN HIMMELFARB, MD**  
*Professor of Medicine*  
*Director, Kidney Research Institute*  
*Co-Director, Center for Dialysis Innovation*  
*Joseph W. Eschbach MD Endowed Chair for Kidney Research*  
*Dept. of Medicine, Div. of Nephrology*  
*University of Washington, Seattle, WA.*

On Thursday, November 15th, 2018, Dr. Jonathan Himmelfarb presented a THRCE seminar that was jointly sponsored by the Department of Medicine titled “Human Kidney on a Chip for Disease Modeling and Toxicity Testing.”

**SUMMARY OF PRESENTATION:**

Drug-induced kidney injury, largely caused by proximal tubular intoxicants, limits development and clinical use of safe and effective drugs. To catalyze the development of drugs that are safe and effective for treating kidney diseases, there is a critical need to be able to model human kidney diseases and injury in vitro during preclinical drug development. The complex multicellular architecture and unusual triad of physiological processes characterized by glomerular filtration, tubular secretion and tubular reabsorption, have often limited the ability of whole organism models to fully recapitulate the diversity and manifestations of human disease. Conventional two-dimensional human epithelial cell models do not accurately recapitulate kidney physiology or disease, and microfluidic flow is essential to kidney nephron structure and function, and is an essential component in recapitulating in vivo physiology and pathophysiology.

We have developed a three dimensional flow directed “kidney-on-a-chip” microphysiological system populated with human kidney cells, which has been extensively tested with functional characterization of key component structures of the proximal tubule and the peritubular microvascular network. We are also able to routinely obtain, isolate and characterize relatively pure primary cultures of multiple
human kidney cell lineages. In addition, we have developed hydrogels consisting of decellularized human kidney cortical extracellular matrix, and demonstrated phenotypic differences when human kidney cells are grown in this matrix. In addition, we have recently incorporated the use of human pluripotent stem cells coupled with gene editing techniques. Our platforms allow for precise control of cellular composition, extracellular matrix, and vascular and tubular geometry and flow. Our goal is to model important human kidney diseases and promote identification of novel safe and effective treatment.

UPCOMING MEETINGS & EVENTS:

- **Southern Regional Meeting of the SSCI/AFMR**
  ~ New Orleans, Louisiana, February 21-23, 2019

- **Distinguished Mayerson-DiLuzio Lectureship**
  ~ Department of Physiology, Tulane University, NOLA, March 11, 2019

- **Tulane WKD Health Screening Fair**
  ~ Lobby of Tulane University Hospital and Clinics, NOLA, March 14, 2019

- **Tulane Health Sciences Research Days Meeting**
  ~ New Orleans, Louisiana, March 18-19, 2019

- **The 2019 Experimental Biology Meeting**
  ~ Orlando, Florida, April 6–9, 2018, 2019
Recent Publications


BOOK PUBLICATIONS:

From September through December 2018, investigators and physicians affiliated with T.H.R.C.E. participated in the following meetings

**AHA Council on Hypertension Sessions (Hypertension/Kidney Council/ASH); Sept. 6-9, 2018; Chicago, IL.**

- Castillo A, Khan N, Shindler I, Navar LG, and Majid DSA. “Inverse Circadian Pattern in the Renal Generation of Tumor Necrosis Factor-Alpha (TNFα) and Angiotensinogen (AGT) in Salt-Sensitive Hypertension (SSH) Induced by Angiotensin II (Ang II) in Mice.”
- Howell NL, Kemp BA, Keller SR, Gildea JJ, Shao W, Navar LG, and Carey RM. “Identification of a Primary Renal AT2 Receptor Defect in Spontaneously Hypertensive Rats (SHR).”
- Kulthinee S, Shao W, Franco M, Navar LG. “Purinergic P2X1 and P2X7 Receptors Activation Attenuate Angiotensin AT1 Receptors Dominance in Regulating the Preglomerular Renal Microcirculation in Angiotensin II Dependent Hypertension.”
- Kumar P, Bloodworth M, Nguyen C, Gogulamudi VR, and Pandey KN. “Differential Regulation of Blood Pressure and Renal Injury by HDAC Inhibitor Mocetinostat in Npr1 Gene-targeted Male and Female Mutant Mice.”
CORE FACILITIES & SERVICES

Tulane Hypertension and Renal Center of Excellence (THRCE) houses 2 research core facilities that were developed during COBRE phases I and II and are now maintained and supported by a COBRE Phase III grant awarded by the NIH/NIGMS. These core facilities are essential for the support of basic, clinical, and translational research in hypertension and renal biology and provide unique research opportunities for emerging leaders by establishing an enriched environment in which to develop investigators in both the clinical and basic hypertension research. The resources and services provided by the Center’s COBRE Core facilities can be utilized by both COBRE and other investigators within Tulane and other institutions for hypertension, cardiovascular and renal research. The 2 research Core facilities are:

- **The Molecular, Imaging, and Analytical Core**: Serves as the resource for instruments and equipment needed to perform advanced molecular biology, semi-quantitative immuno-histochemistry and bio-analytical experiments.
- **Mouse Phenotyping Research Core (MPRC)**: Contains resources to support high-tech data collection capabilities that are unique in the State of Louisiana and essential to research requiring the utilization of an array of methodologies to perform measurements of cardiovascular, blood pressure and renal function in mice.

Other activities of the Center include the sponsorship of local and regional meetings on hypertension and public education programs to increase awareness of the dangers of hypertension. For further information regarding the Core Facilities, please access [http://tulane.edu/som/thrce/core.cfm/](http://tulane.edu/som/thrce/core.cfm/).