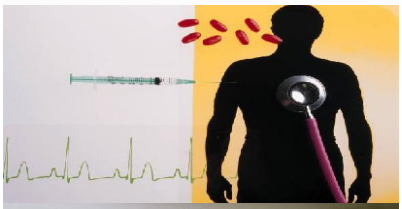
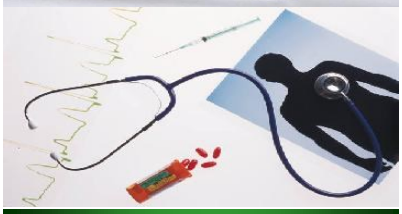




T.H.R.C.E.

TULANE HYPERTENSION AND RENAL CENTER OF EXCELLENCE

Summer 2017 *Volume 16, Issue 2*

In This Issue

AHA/Tulane Research Reception

The American Heart Association (AHA) hosted its annual research reception at Tulane Medical Center on Wednesday, August 16th 2017. The event invited physicians, researchers, the medical community, and AHA donors to see state-of-the-art AHA supported research activities in New Orleans. The AHA invested $2.7 million in funded heart and stroke research in 2016 at several New Orleans institutions. The funds raised are a direct result of the AHA's Go Red Luncheon, AHA Heart Walks and Heart & Soul Gala. Attendees were able to view posters of current AHA sponsored projects and mingle with leading researchers and doctors to learn how AHA sponsored research positively affects local people's lives, their health, their community and their economy.

Tulane faculty, staff, and investigators met AHA donors at the AHA/ Tulane Research Reception that was held August 16th, 2017 at the Tulane Hospital Faculty club. Above are some pictures taken at the reception

Continued...

THRCE to participate in the 2017 American Heart Association Heart Walk

For the last 10 years, representatives of THRCE have participated in the annual American Heart Association (AHA) Heart Walk. The 2017 AHA Heart Walk will be held on November 11th at Champions Square in New Orleans. The Heart Walk is a fun, free, family focused event that promotes exercise and help raise funds. Funds raised in the walk are matched by the AHA's national office and reinvested into the community by funding research, prevention, and treatment of cardiovascular diseases and stroke. Tulane investigators have been the recipients of numerous AHA grants and by the participation of THRCE in AHA Heart walks, we hope to help AHA continue to fund future investigators. Other Tulane representatives participating in the 2017 Heart Walk are the Tulane Heart and Vascular Center and the Departments of Physiology, Pharmacology, and Medicine. Along with the walk, the event include numerous fun-filled health and wellness activities, free food and entertainment. Below are pictures highlighting THRCE activities at past AHA heart walks.

Continued...

The Heart Walk is the American Heart Association's premier event for raising funds to save lives from this country's No. 1 and No. 5 killers - heart disease and stroke. Tulane School of Medicine is committed to support the American Heart Association's mission of building healthier lives free of cardiovascular diseases and stroke. Please support the Tulane teams participating in the AHA Heart Walk by joining them in the walk and by raising lifesaving funds. Money raised gets reinvested back into our local community to fund research and other public education programs. The 2017 Heart Walk will be held on Saturday, November 11th at Champions Square in New Orleans.

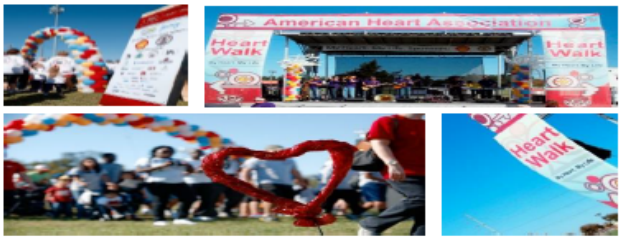
American Heart Association. My Heart. My Life. | Heart Walk

New Orleans Heart Walk

Saturday, November 11, 2017

Champions Square

Festivities Begin 9AM – Walk Begins 10AM



On November 11, 2017, the American Heart Association will host its annual New Orleans Heart Walk at Champions Square.

This is a **FUN, FREE, FAMILY FOCUSED** event that embraces getting active and creating healthier lives free of cardiovascular disease and stroke .

The Heart Walk features:

- Free Food
- Free Drinks
- Music
- Children's Hospital's Kids Heart Challenge Obstacle Course
- T-shirt Design contest and MORE!

2017 Heart Walk Chairperson

Joe Ochipinti
CEO, Gulf State Region
UnitedHealthcare

2017 Heart Walk Executive Leadership Team

Randy Allen
UPS

Lynne Burkart
Cindy Delaparte
Postlethwaite & Netterville

Scott Chapman
UnitedHealthcare

Bill Crombie
USI Insurance.

Greg Feirn
LCMC Health

Kevin Gardner
HUB International

Kyle Godfrey
Tulane University

Vince Gremillion
Restech

Wayne Landwerlin
Arthur J. Gallagher

Gary Lorio
Whitney Bank

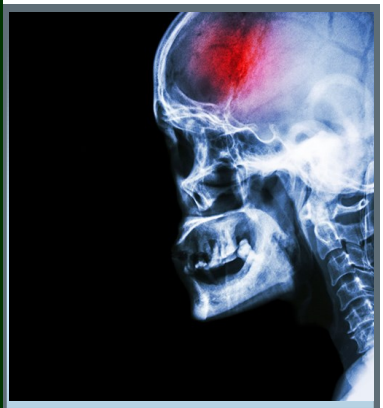
Shirley Naquin
UnitedHealthcare

Brian North
Fifth District Bank

www.neworleansheartwalk.org

Continued…

STUDY COULD LEAD TO BLOOD TEST FOR STROKE DIAGNOSIS



Researchers at Tulane University and Ochsner Medical Center have identified biomarkers in the blood that are a promising target for a potential new test to diagnose a recent ischemic stroke.

Researchers at Tulane University and Ochsner Medical Center have identified biomarkers in the blood that could one day help doctors diagnose a recent ischemic stroke, according to a study published in the August issue of the journal Circulation: Cardiovascular Genetics.

Ischemic strokes, are caused when the contents of plaque in a major artery break away, forming a clot and blocking blood flow to the brain. Quickly identifying whether a patient has had a stroke is critical as clot-busting treatments must be administered within hours to reverse damage and increase odds of a full recovery. No blood test exists for diagnosing stroke or identifying those who are at greatest risk for imminent stroke due to cardiovascular disease.

Ochsner vascular surgeon, Dr. Hernan Bazan, and THRCE affiliate and Assistant Professor of the Department of Physiology at Tulane, Dr. Cooper Woods, compared blood levels of non-coding RNAs, molecules that regulate gene expression, in patients who had a stroke within five days to those who either had not had a stroke or had a stroke more than five days prior. They measured microRNA-221, which helps keep plaque from rupturing by promoting the growth of vascular smooth muscle cells, and circular RNA-284, a suspected inhibitor of microRNA-221. The results showed that an elevation in the ratio of circular RNA-284 to microRNA-221 accurately identified those patients with a recent ischemic stroke.

"Our study offers a promising blueprint for a potential new stroke diagnostic," said Dr. Woods, study co-author. He also added that tools for rapidly measuring RNA in the clinic are being developed, and researchers plan to pair their findings with this emerging technology to develop a point-of-care test.

Further studies are planned to determine if the same non-coding RNA ratio can also predict whether patients are at imminent risk for stroke. "This work represents an important step towards understanding and predicting carotid-related strokes," said Dr. Bazan, Associate Professor of Surgery at Ochsner Health System, "… through ongoing translational research such as this, we are aiming to develop better treatments and work towards preventing these episodes."

GRANTS, HONORS & RECOGNITION AWARDED TO THRCE AFFILIATED INVESTIGATORS

L. Gabriel Navar, PhD:

- Appointed by recommendation of the Council of the American Physiological Society to serve on the Distinguished Physiologists Committee for a three year term.
- The Department of Physiology was awarded the Owl Club's T1 Course of the Year and was nominated for overall Best Department Award.
- Awarded a COBRE No Cost Extension that will support the program through July 2018.
- Gave an invited presentation to the joint Medicine/Physiology seminar titled, "Intratubular Renin-Angiotensin System: A paradigm shift in understanding angiotensin II-Dependent Renal Injury in hypertension & diabetes."

Andrei V. Derbenev, PhD:

- Selected as member of the Tulane Grievance Committee.

Samir El-Dahr, MD:

- Grant Review: NIDDK ZDK1 GRB-3 (O2) Co-Chair: Review Panel, "Developmental Centers in Benign Urology (P20)."
- Beginning August 2017, was awarded a 5 year, NIH/NIDDK RO1 grant for his study titled, "Epigenetic control of Nephron Progenitor Cell Lifespan."
- Was an invited speaker at the Pediatrics Grand Rounds held at University of California San Diego Children's Hospital on June 2017. Presented, "Stem cells for Chronic Kidney Disease."
- On June 27th 2017, gave an invited presentation titled "Epigenetics of Renal Development," at the FASEB Summer Conference on Polycystic Kidney Disease held at Big Sky, Montana.

Keith Ferdinand, MD:

- Tulane cardiologist, Dr. Keith Ferdinand received the 2017 Spirit of the National Heart Leadership Award from the Association of Black Cardiologists on Oct. 7 in New York City.

Kathleen Hering-Smith, PhD:

- Selected as a member of the Tulane University Senate Committee.
- Nominated for the Owl Club's T1 Best PBL Facilitator.

Prerna Kumar, PhD:

- Was a judge at the 21st Annual American Society for Biochemistry and Molecular Biology Undergraduate Poster Competition held in Chicago at the 2017 Experimental Biology (EB) Meeting.
- April 24th 2017, presented a poster at the 2017 EB Meeting titled, "Class I-Specific HDAC Inhibitor Stimulates the Expression of Npr1 in Haplotype Mice by Enhanced Histone Acetylation at Different Lysine Residues."

Norman Kreisman, PhD:

- Received an award for T1 Best PBL Facilitator by the Owl Club and was nominated for the W. Clifford Newman Student Advocacy Award.

Continued...

News

Dewan S. A. Majid, MD. PhD:

- Served as an AHA grant reviewer at the, "AHA Cardio-Renal-Basic" Study Section-2.

Kenneth D. Mitchell, PhD:

- Selected as Committee member of both Tulane Curriculum & Tulane Education Policy.
- Received the Owl Club's Teaching Excellence Award (Inaugural). The Teaching Excellence Award recognizes professors who have taught at least two lectures, and whose excellence in the classroom has achieved an outstanding student rating.

Kailash Pandey, PhD:

- Awarded a 4 year, competing renewal NIH/NHLBI R01 grant beginning April 2017 for his research, "Study of ANP Receptor: Gene-Targeting and Expression."
- Appointed to the Editorial Board of Physiological Genomics and the Editorial Board of Molecular and Cellular Biochemistry.

Minolfa Prieto, PhD:

- Appointed to the Hypertension and Microcirculation Study Section.
- Elected as committee member of the Tulane Faculty Tenure, Freedom, and Responsibility.
- Invited to serve as a Member of the KCVD Leadership Committee of the "Council on The Kidney in Cardiovascular Disease "beginning July 1 2017.
- Nominated for the Owl Club's T1 Best PBL Facilitator.

T. Cooper Woods, PhD:

- Received funding from the Clinical Research & Innovation Support Program (CRISP) at Ochsner. The title of his project is "Mapping serum biomarkers of carotid plaque rupture to intra-plaque changes: Novel predictors of stroke."
- Received the Owl Club's Teaching Excellence Award (Inaugural) and was nominated for T1 Best PBL Facilitator.

Zubaida Saifudeen, PhD:

- Selected as members of the Tulane Grievance Committee.

Students & Post-doctoral fellows:

Post-doctoral fellows

- **Kulthinee, Supaporn, (Tom) PhD** (Mentor: Dr. L. Gabriel Navar)
  - ◊ Received the John F. Perkins, Jr. Memorial Award for International Physiologists from the American Physiological Society (APS) on June 9, 2017.

Gradate & Medical Students

- **Donald Wathieu**, medical student (Mentor: Dr. TC Woods)
  - ◊ Received a 2017 Student Scholarship in Cardiovascular Disease. The title of his project is "The role of microRNAs in Stimulating pro-inflammatory polarization of macrophages in Diabetes."

News

2017 SUMMER STUDENTS WELCOMED TO THRCE

Each year meritorious Medical and Undergraduate Research Students are selected to work with faculty researchers affiliated with the Tulane Hypertension & Renal Center of Excellence for 8 to 10 weeks during the summer. Each student receives a stipend, and is exposed to the valuable nature of a career path in research, and has the opportunity to attend the various THRCE events and Seminars. The following students were selected for the 2017 Summer Research Program:

MEDICAL STUDENTS:

Sponsor: Bourgeois Medical Research Endowment

- **Neelesh Jain**
  Mentor: Dr. Kenneth D. Mitchell

DeBakey Scholar

- ***Donald Wathieu***
  Mentor: Dr. T. Cooper Woods

Sponsor: AHA Summer Fellowship Program

- **Matthew Hennrikus**
  Mentor: Dr. Minolfa C. Prieto
- **Thomas John**
  Mentor: Dr. Minolfa C. Prieto
- ***Michael Kremer***
  Mentor: Dr. T. Cooper Woods

UNDERGRADUATE STUDENTS:

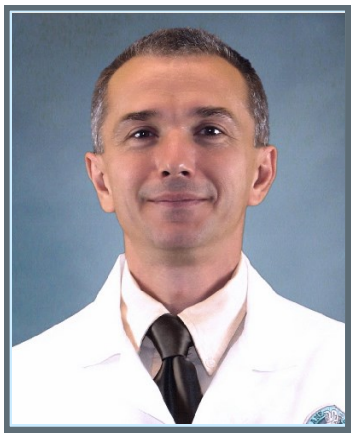
- **Marco Acosta (Volunteer)**
  Mentor: Dr. Minolfa C. Prieto
- **Justine Jorgensen (Tulane)**
  Mentor: Dr. Minolfa C. Prieto
- **Emily B. Kaminski**
  Mentor: Dr. A. V. Derbenev
- **Justin Mourain (LSU)**
  Mentor: Dr. Minolfa C. Prieto
- **Christopher Wong**
  Mentor: Dr. Minolfa C. Prieto

Louisiana Biomedical Research Network-LSU

- ***Gaurav Phuyal***
  Mentor: Dr. Dewan SA Majid

THRCE SPONSOR
LOCAL, NATIONAL & INTERNATIONAL SPEAKERS

THRCE sponsors bi-weekly seminars by scheduling local as well as nationally and internationally recognized investigators and clinicians in the field of hypertension research, treatment and education. Speakers who present at the THRCE Seminar Series are asked to provide a brief summary of their talk that we can share with our newsletter audience. From May through August, 2017, the following speakers presented THRCE seminars.



- **IHOR V. YOSYPIV, MD**
  Associate Professor of Pediatrics,
  Department of Pediatrics,
  Chief, Division of Pediatric Nephrology,
  Tulane University School of Medicine,
  New Orleans, LA.

On May 18, 2017, Dr. Yosypiv presented a THRCE seminar titled, "Foxd1 is an upstream regulator of the renin-angiotensin system during metanephric kidney development."

SUMMARY:

Congenital Anomalies of the Kidney and Urinary Tract (CAKUT) account for a majority of children with end-stage-renal disease requiring dialysis and renal transplantation. All form of CAKUT stem from abnormal kidney development due to aberrant interactions between the ureteric bud (UB) and mesenchyme. We tested the hypothesis that Foxd1, a transcription factor essential for normal kidney development, is an upstream regulator of the renin-angiotensin system (RAS) during UB branching morphogenesis in mouse model. By embryonic day E12.5, the number of UB tips was reduced in Foxd1-/- compared to Foxd1+/+ metanephroi. Quantitative RT-PCR demonstrated that renin, angiotensin I-converting enzyme (ACE), angiotensin (Ang) II receptor type 1 (AT1R) mRNA levels were decreased in

Continued...

Foxd1-/- compared with Foxd1+/+ E14.5 metanephroi. Western blot analysis and immunohistochemistry showed decreased expression of angiotensinogen (AGT) and renin proteins in Foxd1-/- compared with Foxd1+/+ metanephroi. Foxd1 overexpression in mesenchymal MK4 cells in vitro increased renin, AGT, ACE and AT1R mRNA levels. Exogenous Ang II stimulated UB branching equally in whole intact E12.5 Foxd1-/- and Foxd1+/+ metanephroi grown ex vivo. We conclude that Foxd1 is an upstream positive regulator of the RAS during early metanephric development and propose that the cross-talk between Foxd1 and RAS is essential in UB branching morphogenesis.

Congenital reduction of nephron number, a condition called RHD, is associated with subsequent hypertension and chronic kidney disease in humans. Dr. Yosypiv's laboratory is interested in the basic mechanisms which control UB branching morphogenesis and nephrogenesis, and the role of the renin-angiotensin system (RAS) gene mutations in human CAKUT. We examined cytogenomic aberrations and performed Sanger sequencing of key RAS genes in children with multicystic dysplastic kidney (MCDK) and performed Sanger sequencing of key RAS genes in children with other forms of CAKUT. We identified novel associations of mutations in the genes encoding renin, AGT, ACE or AT1R with isolated MCDK, and of the prorenin receptor (PRR) mutations with other forms of CAKUT in children in the United States. These findings highlight the crucial role of the RAS in the pathogenesis of MCDK and CAKUT in children and may help develop novel therapies that can be applied to the study of nephron regeneration strategies in CAKUT.

- **VECIHI BATUMAN, MD, FACP, FASN**
  Professor, Department of Medicine,
  Section of Nephrology & Hypertension,
  Director, Medicine Service Line, SLVHCS, New Orleans,
  Tulane University School of Medicine, New Orleans, LA.

Dr. Vecihi Batuman presented, "Kidney Injury From Paraproteins - The role of Inflammatory Pathways" on June 15, 2017.

Continued...

SUMMARY:

Dr. Batuman presented his research on the mechanisms of acute and chronic kidney disease in multiple myeloma. The data showed that immune globulin light chains mediate adverse effects on the kidney beyond cast formation and that they inhibit substrate transport, induce cell stress responses including production and release of inflammatory cytokines, and induce epithelial-to-mesenchymal transition of renal proximal tubule cells, in vitro. Light chain-induced cytokines contribute to the interstitial inflammatory changes often seen in myeloma associated kidney disease—possibly a more important mechanism of kidney injury than cast formation. The light chain-induced renal injury is mediated by inflammatory pathways signaled through MAPKs, particularly p38 MAPK and through activation of nuclear factor- kappa B. The cytotoxic effects of immunoglobulin light chains on proximal tubule cells require endocytosis and blocking endocytosis abrogates renal injury, which also can potentially be exploited as a therapeutic maneuver. Light chain induced inflammatory and proinflammatory cytokines and epithelial-mesenchymal transition contribute to both acute and chronic kidney injury associated with multiple myeloma and other monoclonal gammopathies. Current work in Dr. Batuman's laboratory suggest that light chains trigger inflammatory pathways in the kidney through activation of innate immunity mediated by Toll-like receptors, TLR 2, 4, and 6. Identifying the inflammatory pathways is likely to also identify potential therapy opportunities that may help improve the clinical outcomes in myeloma patients with kidney involvement.

- **RYOSUKE SATO, PHD**
  Assistant Professor, Department of Physiology,
  Director, Molecular Core Facility,
  Tulane University School of Medicine, New Orleans, LA.

THRCE Molecular Facility Director, Dr. Ryosuke Sato, presented the THRCE seminar, "Intrarenal angiotensinogen regulation in diabetes mellitus and the effects of an SGLT2 inhibitor" on July 13, 2017.

Continued...

SUMMARY:

Type 2 diabetes mellitus is a complex disease where hyperglycemia occurs as a result of the development of insulin resistance. It is often associated with obesity and is characterized by hyperglycemia, hyperinsulinemia, as well as hyperleptinemia. In addition to the problems associated with poor glucose control, type-2 diabetes is accompanied by a chronic inflammatory state. This chronic inflammatory state leads to a number of associated complications, including cardiovascular and renal diseases. In addition to established traditional treatment options, one new approach has focused on blocking glucose reabsorption by inhibiting SGLT2 in renal tubules to allow substantial excretion of glucose in the urine. A key factor in the intrarenal renin-angiotensin syetm activation is stimulation of intrarenal angiotensinogen which is the precursor of angiotensin peptides. Therefore, we tested effects of an SGLT2 inhibitor on intrarenal angiotensinogen regulation. In the seminar, I presented experimental results obtained from animal and cell studies in the project.

- **DEWAN S.A. MAJID, MD, PHD**
  Professor, Department of Physiology,
  Director, Mouse Phenotype Core Facility,
  Tulane University School of Medicine, New Orleans, LA.

On July 27 2017, Dr. Dewan Majid, Director of THRCE Mouse Phenotype Facility, presented, "Evidence for pro-hypertensive effect of Interleukin-10 in Angiotensin II induced salt-sensitivity."

SUMMARY:

Interleukin-10 (IL-10; generally known as anti-inflammatory cytokine) has been suggested to play a protective role in cardiovascular disorders induced by angiotensin II (AngII) and other factors that are associated with inflammation. In this talk, the overall role of IL-10 in the regulation of kidney function as well as in the development of salt-sensitive hypertension (SSH) has been presented. Recent studies in our laboratory (Singh et al-2014; Physiol Rep. 2:e00228) have demonstrated that IL-10 is normally present in plasma and its protein is

Continued...

constitutively expressed in the renal tissue mostly in epithelial cells of distal tubular segments in the kidney. Renal tissue expression of IL-10 was shown to be regulated by nitric oxide (NO). Reduction of NO formation leads to decrease in IL-10 protein immune-expression in the kidney which can be restored by supplementing NO by NO donors. Suppression of IL-10 by reduction in NO formation also enhance protein expression of pro-inflammatory cytokine, tumor necrosis factor-alpha (TNF-α). We have evaluated the renal effects of IL-10 administration in mice, changes in IL-10 levels in plasma and kidney tissues in mice during chronic intake of high salt (HS) containing diets and most importantly the contributing role of this cytokine in the development of SSH induced by chronic administration of AngII. These data suggest that IL-10 generally exerts vasodilator and hyper-filtration effects in the kidney but minimally influences the renal excretory function. Chronic HS intake generally suppresses IL-10 level, particularly in the kidney. To examine its role in SSH induced by AngII, we have recently performed the experiments to evaluate the responses to chronic AngII (400 ng/min/kg bw; osmotic minipump) infusion in IL-10 gene knockout (IL-10KO) mice, fed with either normal (NS; 0.3% NaCl) or high (HS; 4% NaCl) salt diets and these responses were compared to those in wild type (WT) mice (Singh et al -2017;Hypertension.70:839-845). NS or HS diets were given alone for the first 2 weeks and then with AngII treatment for additional 2 weeks. Arterial pressure was continuously monitored by implanted radio-telemetry and 24 hour urine collection was performed by metabolic cages on the last day of the experimental period. Basal mean arterial pressure (MAP) was lower in IL-10KO than in WT mice. MAP responses to NS/HS alone or to the AngII+NS treatment were similar in both strains. However, the increase in MAP induced by the AngII+HS treatment was significantly lower in IL-10KO compared to WT. Renal tissue eNOS expression as well as urinary excretion of nitric oxide (NO) metabolites, nitrate/nitrite were higher in IL-10KO compared to WT. These results indicate that an increase in NO production helps to mitigate SSH induced by AngII and suggest that a compensatory interaction between IL-10 and NO exists in modulating AngII induced responses during HS intake. IL-10 deficiency minimizes hypertensive response induced by chronic AngII + HS intake. This is a novel finding as many previous studies implicated the opposite notion that a decrease in IL-10 may propagate AngII induced cardiovascular dysfunction. The implied pro-hypertensive role of IL-10 in SSH induced by AngII would have significant impact on the therapeutic approaches in the management of inflammatory renal in-jury associated with many hypertensive conditions.

- **FEDERICO J. TERAN, MD**
  Nephrologist, Department of Nephrology,
  Section of Nephrology and Hypertension,
  Tulane University School of Medicine, New Orleans, LA.

August 24th THRCE seminar, "Evidence for pro-hypertensive effect of Interleukin-10 in Angiotensin II induced salt-sensitivity" was presented by Dr. Federico Teran.

SUMMARY:

More than half of all filtered elements are reabsorbed in the proximal tubules of the kidneys. Transporters such as the sodium dicarboxylate 1 (NaDC1) play a vital role in the transport of Kreb cycle intermediates. The tubular effect of some of these intermediates, such as citrate's role in kidney stone prevention, has been well described. Yet the tubular properties of α-ketoglutarate (α-KG) and Succinate are not fully known. Recent data has implicated α-KG, as a paracrine mediator, in the acid base homeostasis of the distal nephron by increasing tubular bicarbonate. Similarly, it has been claimed that tubular Succinate can contribute to development of hypertension by stimulating renin release.

In this series, we used a knockout mouse model of NaDC1 on three different diets, normal, acid, and alkali rich diets to elucidate the urinary effects of tubular α-KG and Succinate. Urinary α-KG and Succinate levels were significantly increased in the knockout mouse (compared to wild type). There were notable urinary pH changes based on diet and genotype but not to the degree we expected. Likewise, although, tubular Succinate was elevated in the knockout mouse, no significant difference in blood pressure was noted between the groups. We also took advantage of sex differences as females tend to have higher Succinate levels but again, no difference in blood pressure was appreciated between the sexes. One familiar finding was the reduced mRNA expression of the downstream receptors for the respective α-KG and Succinate. This possible adaptive mechanism could explain the differences that maintained the pH within a narrow range and maintained blood pressure normal. Better characterization of these novel interactions is necessary as polymorphism in these pathways may account for individual variances that can result in pathological states.

Publications

Recent Publications (includes those omitted from previous newsletters)

- **Bazan HA, Hatfield SA, Brug A, Brooks AJ, Lightell DJ Jr, Woods TC.** Carotid Plaque Rupture Is Accompanied by an Increase in the Ratio of Serum circR-284 to miR-221 Levels. *Circ Cardiovasc Genet. 2017 Aug;10(4). pii: e001720. doi: 10.1161/CIRCGENETICS.117.001720. PMID: 28779016.*
- **Bourgeois CT, Satou R, Prieto MC**. HDAC9 is an epigenetic repressor of kidney angiotensinogen establishing a sex difference. *Biol Sex Differ. 2017 May 30;8:18. doi: 10.1186/s13293-017-0140-z. eCollection 2017. PMID: 28572913, PMCID: PMC5450130.*
- **El-Dahr SS, Li Y, Liu J, Gutierrez E, Hering-Smith KS, Signoretti S, Pignon JC, Sinha S, Saifudeen Z.** p63+ ureteric bud tip cells are progenitors of intercalated cells. *JCI Insight. 2017 May 4;2(9). pii: 89996. doi: 10.1172/jci.insight.89996. [Epub ahead of print]. PMID: 28469077. PMCID: PMC5414549.*
- **Franco M, Bautista-Pérez R, Cano-Martínez A, Pacheco U, Santamaría J, Del Valle Mondragón L, Pérez-Méndez O, Navar LG**. Physiopathological implications of P2X1 and P2X7 receptors in regulation of glomerular hemodynamics in angiotensin II-induced hypertension. *Am J Physiol Renal Physiol. 2017 Jul 1;313(1):F9-F19. doi: 10.1152/ajprenal.00663.2016. Epub 2017 Apr 12. PMID: 28404593.*
- **Gonzalez AA, Lara LS, Prieto MC.** Role of Collecting Duct Renin in the Pathogenesis of Hypertension. *Curr Hypertens Rep. 2017 Aug;19(8):62. doi: 10.1007/s11906-017-0763-9. Review. PMID: 28695400.*
- **Gonzalez AA, Salinas-Parra N, Leach D, Navar LG, Prieto MC.** PGE2 upregulates renin through E-prostanoid receptor 1 via PKC/cAMP/CREB pathway in M-1 Cells. *Am J Physiol Renal Physiol. 2017 Jul 12:ajprenal.00194.2017. doi: 10.1152/ajprenal.00194.2017. [Epub ahead of print]. PMID: 28701311*
- **Gonzalez AA, Zamora L, Reyes-Martinez C, Salinas-Parra N, Roldan N, Cuevas CA, Figueroa S, Gonzalez-Vergara A, Prieto MC.** (Pro)renin receptor activation increases profibrotic markers and fibroblast-like phenotype through MAPK-dependent ROS formation in mouse renal collecting duct cells. *Clin Exp Pharmacol Physiol. 2017 Jul 11. doi: 10.1111/1440-1681.12813. [Epub ahead of print]. PMID: 28696542.*
- **Kobayashi H, Liu J, Urrutia AA, Burmakin M, Ishii K, Rajan M, Davidoff O, Saifudeen Z, Haase VH.** Hypoxia-inducible factor prolyl-4-hydroxylation in FOXD1 lineage cells is essential for normal kidney development. *Kidney Int. 2017 Aug 26. pii: S0085-2538(17)30475-1. doi: 10.1016/j.kint.2017.06.015. [Epub ahead of print]. PMID: 28847650.*
- **Liu J, Edgington-Giordano F, Dugas C, Abrams A, Katakam P, Satou R, Saifudeen Z.** Regulation of Nephron Progenitor Cell Self-Renewal by Intermediary Metabolism. *J Am Soc Nephrol. 2017 Jul 28. pii: ASN.2016111246. doi: 10.1681/ASN.2016111246. [Epub ahead of print]. PMID: 28754792*
- **Mehaffey E, Majid DSA**. Tumor necrosis factor-alpha, Kidney function and hypertension. *Am J Physiol Renal Physiol. 2017 Jul 19:ajprenal.00535.2016. doi: 10.1152/ajprenal.00535.2016. [Epub ahead of print].* PMID: 28724611 DOI: 10.1152/ajprenal.00535.2016.

Publications

Continued...

- **Pingili AK, Davidge KN, Thirunavukkarasu S, Khan NS, Katsurada A, Majid DSA, Gonzalez FJ, Navar LG, Malik KU**. 2-Methoxyestradiol Reduces Angiotensin II-Induced Hypertension and Renal Dysfunction in Ovariectomized Female and Intact Male Mice. *Hypertension. 2017 Jun;69(6):1104-1112. doi: 10.1161/HYPERTENSIONAHA.117.09175. Epub 2017 Apr 17. PMID: 28416584, PMCID: PMC5426976, DOI: 10.1161/HYPERTENSIONAHA.117.09175.*
- **Prieto MC, Reverte V, Mamenko M, Kuczeriska M, Veiras LC, Rosales CB, McLellan M, Gentile O, Jensen VB, Ichihara A, McDonough AA, Pochynyuk OM, Gonzalez AA.** Collecting Duct Prorenin Receptor Knockout Reduces Renal Function, Increases Na+ Excretion and Mitigates renal Responses in ANGII induced hypertensive mice. *Am J Physiol Renal Physiol. 2017 Aug 16:ajprenal.00152.2017. doi: 10.1152/ajprenal.00152.2017. [Epub ahead of print]. PMID: 28814438.*
- **Rosivall L, Cypress M, Navar LG**. Editorial. *Physiol Int. 2017 Jun 1;104(2):91-96. doi: 10.1556/2060.104.2017.2.9. No abstract available. PMID: 28665195*
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- **Song R, Lopez MLSS, Yosypiv IV.** Foxd1 is an upstream regulator of the renin-angiotensin system during metanephric kidney development. *Pediatr Res. 2017 Aug 2. doi: 10.1038/pr.2017.157. [Epub ahead of print].* PMID: 28665931 DOI: 10.1038/pr.2017.157

Calendar of Events

THRCE Seminars

September 21

SARAH LINDSEY, PHD
Assistant Professor, Department of Pharmacology,
Tulane University School of Medicine, New Orleans, LA.
"Estrogen Receptor Signaling in Arterial Stiffness."

October 5

KENNETH D. MITCHELL, PHD, FAHA, FASN
Professor, Department of Physiology,
Director, Medical Education,
Director, DeBakey Scholars Program,
Tulane University School of Medicine, New Orleans, LA.
"ANG II-Dependent Hypertension: An Unexpected Journey."

October 12 **

DAVID W. PLOTH, MD
Distinguished Professor, Endowed Chair: Williams,
Department of Medicine, Division f Nephrology,
Medical University of South Carolina, Charleston, SC.
"Unexpected Prevalence of CKD, Diabetes, and Hypertension in Rural Tanzania."

October 19

PRERNA KUMAR, PHD
Instructor, Department of Physiology,
Tulane University School of Medicine, New Orleans, LA.
"Sodium Butyrate & Retinoic acid Attenuate Renal Inflammation & Fibrosis in Npr1 Haplotype Mice."

November 2

EDGAR A. JAIMES, MD
Chief of Renal Service, Department of Medicine,
Memorial Sloan Kettering Cancer Center,
Professor of Medicine, Weill Cornell Medical College,
Memorial Sloan Kettering Cancer Center, New York, NY.
"Hypertension, Renal Disease and Cancer."

November 3**

AKIRA NISHIYAMA, MD, PHD
Chairman & Professor, Department of Pharmacology,
Kagawa University Medical School, Kagawa, Japan.
"(pro)renin receptor as a therapeutic target of cancer."

November 16

JING CHEN, MD, MMSC, MSC
Associate Professor of Medicine, Department of Medicine,
Division of Nephrology and Hypertension,
Tulane University School of Medicine, New Orleans, LA.
Talk: TBA

November 30

THOMAS COOPER WOODS, PHD
Assistant Professor, Department of Physiology & Tulane Heart & Vascular Institute,
Tulane University School of Medicine, New Orleans, LA.
Talk: TBA

January 11, 2018

HONGBING LIU, PHD
Assistant Professor, Department of Pediatrics,
Tulane University School of Medicine, New Orleans, LA.
"Intrauterine growth restriction (IUGR) and kidney development."

Conferences are held alternative Thursdays at 4:00pm in the Tulane Medical School, Pharmacology Library, Room 4700
*** Denotes the seminar date is not our normally scheduled day.*

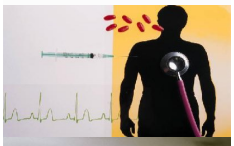
CORE FACILITIES & SERVICES

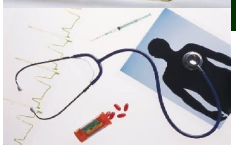
Tulane Hypertension and Renal Center of Excellence (THRCE) houses 4 research core facilities that were developed during COBRE phases I and II and are now maintained and supported by a COBRE Phase III grant awarded by the NIH/NIGMS. These core facilities are essential for the support of basic, clinical, 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 4 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.
- **Animal and Gene-Targeted Core:** Maintains and generates new breeding pairs, performs genotyping, and maintains colonies of genetically manipulated mice and rats.
- **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.
- **Clinical and Translational Core:** Promotes and facilitates clinical and translational studies in hypertension, kidney disorders, and related cardiovascular diseases.

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/**

T.H.R.C.E.





Tulane Hypertension & Renal Center of Excellence (THRCE) will appreciate any support for the continual development of the center and its CORE Facilities, the publication of the THRCE newsletters, and the support of the THRCE bi-weekly seminars series. All donations to the center and its activities are considered tax-deductible.

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Comments are welcome:
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The directors invite faculty members interested in participating in the activities of the T.H.R.C.E. to submit your name, phone number, fax number, and e-mail address to the Senior Administrative Program Coordinator, Nina R. Majid, by e-mail at htnctr@tulane.edu or regular mail to the address provided. Also, please forward all information (awards, publications, presentations and other news items) to this email address for inclusion in the next newsletter.