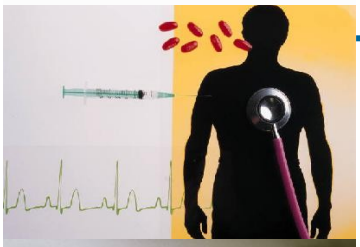
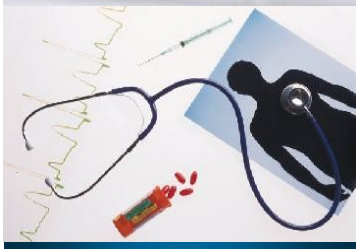




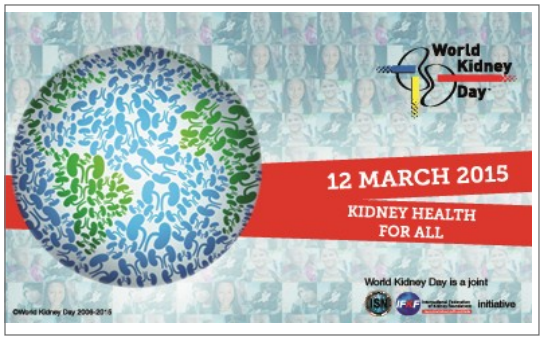
T.H.R.C.E.

TULANE HYPERTENSION AND RENAL CENTER OF EXCELLENCE

Winter 2014 *Volume 13, Issue 3*

In This Issue

MARCH 12, 2015 IS WORLD KIDNEY DAY!



"***Kidney Health for All***" is the theme for the 2015 World Kidney Day (WKD). Within both higher and lower income countries there are communities that are at greater risk than others because of their ethnic origin, socioeconomic status and/or where they live. WKD is a global health campaign that aims to raise awareness of the importance of our kidneys to our overall health, and to reduce the frequency and impact of kidney disease and its associated health problems. Taking steps to live a healthy lifestyle clearly helps to reduce risk, and early detection and treatment can slow or prevent the progression of Chronic Kidney Disease (CKD), and reduce the increased incidence of associated cardiovascular disease. WKD is a joint initiative of the International Society of Nephrology (ISN) and the International Federation of Kidney Foundations (IFKF). More information on WKD can be accessed at: *http://www.worldkidneyday.org/#sthash.G9pW6Ivp.dpuf.*

To commemorate 2015 WKD, THRCE will co-host two special seminars by Dr. David G. Harrison, Professor of Medicine & Pharmacology at Vanderbilt University. THRCE and the Department of Medicine, Division of Nephrology, will jointly sponsor a special WKD "Medicine Grand Rounds" lecture on March 11th, 2015; the title of the talk will be, "The History of Hypertension and the Mosaic Theory." A second special WKD seminar titled, "Inflammation, Immunity and Hypertension" will be held the following day, on March 12th, 2015; that presentation will be jointly sponsored by both THRCE and the Department of Physiology. For further information on the two special WKD seminars, please refer to the "Calendar of Events" section on page 12.

News

HONORS & RECOGNITION AWARDED TO THRCE AFFILIATED INVESTIGATORS

L. Gabriel Navar, PhD:

- Awarded the E. Eric Muirhead Lectureship on November 5th, 2014 at the University of Tennessee Health Science Center in Memphis, Tennessee.
- Notified that his outstanding achievements has earned him a position in Who's Who in America 2015 (69th edition).

Andrei Derbenev, PhD:

- Selected as Member of the advisory committee for Tulane Neuroscience Program/Brain Institute.

Kathleen Hering-Smith, PhD:

- Selected as ASN Kidney STARS mentor.
- Invited to attend the NIH/NIDDK New PI workshop at Bethesda, Maryland on December 2nd and 3rd, 2014.

Dewan S. A. Majid, MD, PhD:

- Invited to attend the 4th Biennial Conference of South Asian Association of Physiologists (SAAP) held in Dhaka, Bangladesh from December 5th to 7th where he Chaired the Plenary Session on "Heavy Metal Toxicity and Oxidative Stress."
- Invited to visit the National Institutes of Cardio-Vascular Diseases (NICVD) in Dhaka, Bangladesh where he presented a seminar and met with physicians, faculties, and post-graduate trainee doctors.
- Invited to Ibne Sina Medical College in Dhaka, Bangladesh where he met with faculties and M.Phil students in the Basic Sciences Departments and presented a lecture.
- Invited to serve on the "Cardiorenal" Study Section of AHA South East affiliate.
- Notified that his outstanding achievements has earned him a position in Who's Who in America 2015 (69th edition).

Kailash N. Pandey, PhD:

- Nominated to Marquis Who's Who.

Minolfa C. Prieto, MD, PhD:

- Awarded a 5 year R01 research grant from the National Institutes of Health, NIDDKD section to study "Pleiotropic effects of Prorenin receptor in collecting duct and intrarenal RAS activation."
- Invited as Special Visiting Professor by Federal University of Rio de Janeiro, Brazil. July 9 through August 31. This activity was part of her commitment with

Honors & Recognition Awarded to THRCE Affiliated Investigators, continued...

Science Without Boarders Program grant of which she is Co-Investigator. On August 2nd she presented a poster titled, "Hyperglycemia induces the apical insertion of the prorenin receptor in the collecting duct cells."

- Promoted to Associate Professor with tenure in December 2014.
- Selected as Reviewer for the Hemodynamic Microcirculation (HM) Study Section of the NIH
- Selected as Member of the Steering Committee of the Physiological Group of the APS-Newsletter, and elected as Editor Chair on October 2014.
- Selected as Member of the Awards Committee of the American Physiological Society in December 2014.

Zubaida Saifudeen, PhD:

- Awarded by the NIDDK-sponsored Diabetic Complications Consortium (DiaComp) a one year NIH/NIDDK DiaComp Pilot & Feasibility Project for her study, "p53-Regulated Metabolic Fitness of Self-Renewing Nephron Progenitor Cells."

Ryosuke Sato, PhD:

- Promoted to Assistant Professor on January 1, 2015.

T. Cooper Woods, PhD :

- Received one year pharmaceutical grant from ACell, Inc. titled, "Effect of MatriStem on cell behavior in the context of diabetic wound healing."
- Received a UQ-Ochsner Seed Fund for Collaborative Research-Bazan, titled: "Alteration of the transcriptome during acute carotid atherosclerotic plaque rupture, Year 2 Study." The subcontract is for $50,000.

Andrea Zsombok, PhD:

- Awarded a subcontract with LSUHSC, in which she is a collaborator on a R01 grant awarded to Dr. Eric Lazartigues.
- In October 2014 participated in an NIH Study Section (Neuroendocrinology, Neuroimmumology, Rhythms and Sleep) held in Baltimore, Maryland as an adhoc reviewer.
- Selected as Member of the advisory committee for Tulane Neuroscience Program/Brain Institute.
- Awarded funding for a scientific research proposal for the Engaged Learning and teaching (CELT). The award of $2,000 will cover the project of undergraduate student, Courtney Nugent.

News

THRCE SPONSORED LOCAL, NATIONAL & INTERNATIONAL SPEAKERS

THRCE sponsors bi-weekly seminars by scheduling nationally and internationally recognized investigators and clinicians in the field of hypertension research, treatment and education. From September through December 2014, the center hosted the following speakers to present THRCE seminars:

- **Sean P. Didion, PhD**
  Associate Professor and Director, MD/PhD Program
  Department of Pharmacology and Toxicology
  Department of Neurology, Cardiovascular-Renal Research Center
  The University of Mississippi Medical Center.

On Thursday, September 25th, 2014, Dr. Sean Didion presented a talk jointly sponsored by the Department of Physiology and THRCE. The talk he gave was entitled "Inflammation, Endothelial Dysfunction, and Angiotensin Too."

Summary: Angiotensin II is associated with vascular hypertrophy, endothelial dysfunction and the activation of a number of inflammatory molecules, however the spatial-temporal relationship involved in the development of hypertension and endothelial dysfunction produced in response to Angiotensin II is not well defined. Inflammation is a key element of vascular disease and understanding the molecular mechanisms that contribute to the impairment of endothelial function is clinically important, especially as carotid artery disease increases markedly with age and is a major contributing risk factor for ischemic stoke and cognitive impairment. Based on studies utilizing genetically altered interleukin-6, interleukin-10, endothelial nitric oxide, and NADPH oxidase (Nox2) mice, Dr. Didion's laboratory has been able to provide important roles for these gene products in the temporal and spatial development of endothelial dysfunction in hypertension as well that which occurs with obesity and normal aging. With a much more definitive understanding of inflammatory gene products in the development of endothelial dysfunction, efforts can now be focused on identifying pharmacological modalities which can be used to limit Inflammation and improve endothelial function in human hypertension. In addition to pharmacological approaches, studies in Dr. Didion's laboratory examine the genome-wide changes in vascular gene expression that occur in Angiotensin II-induced hypertension, which could potentially be used to develop therapies to intervene at critical phase of hypertension development.

- **Kenneth D. Mitchell, PhD**
  Professor, Department of Physiology,
  Tulane University School of Medicine,
  New Orleans, LA.

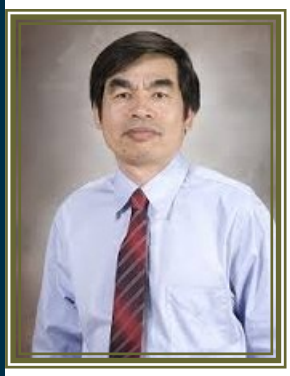
On October 23rd, Dr. Kenneth D. Mitchell, Senior COBRE Mentor, presented a seminar entitled "Renal Derangements in ANG II-Dependent Hypertension: Role of PDGF."

Summary: Studies performed in Dr. Mitchell's laboratory have demonstrated that the renal morphological changes that occur in Cyp1a1-Ren2 transgenic rats with ANG II-dependent malignant hypertension are characterized by increased proliferating cell number in cortical tubules and cortical interstitium, and increased collagen deposition in the renal interstitium. Such renal pathological changes involve activation of PDGF receptor-related kinase, and blocking this pathway ameliorates the renal morphological abnormalities observed in this model of ANG II-dependent malignant hypertension. In addition, chronic PDGF receptor antagonism with imatinib mesylate improves renal hemodynamics independent of changes in blood pressure in Cyp1a1-Ren2 rats with ANG II-dependent malignant hypertension. Furthermore, it was demonstrated that acute blockade of PDGF receptors elicits decreases in mean arterial blood pressure and renal vascular resistance in Cyp1a1-Ren2 transgenic rats with malignant hypertension. These data indicate that activation of PDGF receptors plays an important role in maintaining the elevated arterial blood pressure and renal vascular resistance that occur in ANG II-dependent malignant hypertension. More recent studies have shown that Cyp1a1-Ren2 rats induced with indole-3-carbinol (I3C) also exhibited increases in PDGFβ protein levels in both the renal cortex and renal medulla, elevated PDGFβ receptor levels in both renal cortex and renal medulla, marked proteinuria and elevated urinary ANG II excretion. Chronic PDGF receptor blockade with imatinib did not alter the magnitude of the I3C-induced increase in systolic blood pressure but prevented the increase in urinary ANG II excretion and the increases in PDGFβ protein levels in both renal cortical and medullary tissue. Collectively, the data presented indicate that elevated levels of PDGFβ protein and PDGFβ receptors contribute importantly to the renal injury, the derangements in renal hemodynamics and the increased urinary protein and ANG II levels in ANG II-dependent malignant hypertension.

- **Aaron S. Dumont, MD**
  Charles B. Wilson Professor & Chairman,
  Department of Neurosurgery,
  Tulane University School of Medicine,
  New Orleans, LA.

Dr. Dumont presented, "Inflammation and Cerebral Aneurysms," at the November 6th THRCE Seminar.

Summary: Cerebral aneurysms remain a devastating disease. Rupture of cerebral aneurysms produces subarachnoid hemorrhage which is associated with high morbidity and mortality. Moreover, our treatment options for these dangerous lesions remain suboptimal and there is no currently available medical therapy. Inflammation appears to be central in the development, progression and rupture of cerebral aneurysms, yet our understanding of the underlying mechanisms remains incomplete. TNFalpha is an important inflammatory modulator and is known to play important roles in other human diseases including rheumatoid arthritis. In cerebral aneurysms, TNFalpha appears to alter vessel wall homeostasis and specifically appears to induce a pro-inflammatory, pro-matrix remodeling phenotype in vascular smooth muscle cells. This subsequently perpetuates the inflammatory response, degrades the vessel wall and leads to cell death which ultimately may promote cerebral aneurysm formation, progression and rupture. Using in vitro and in vivo models, data supporting the role of TNFalpha in cerebral aneurysm biology is reviewed and TNFalpha is highlighted as a potential future therapeutic target for patients harboring these lesions.



- **Wenzheng Zhang, PhD**
  Associate Professor, Department of Internal Medicine,
  Division of Renal Diseases & Hypertension,
  University of Texas Medical School,
  Houston, TX.

Dr. Wenzheng Zhang presented, "Epigenetics of Blood Pressure Control and Aqp2+ Renal Progenitor Cells," at the November 20th THRCE Seminar.

Continued...

Summary: The collecting duct system of the kidney contains a series of tubules and ducts that connect the nephrons to the ureter. It is the last part of the tubules that maintain the body's electrolyte/fluid and acid-base balance through the altered function in principal cells and intercalated cells, respectively. The principal cells reabsorb sodium via the epithelial sodium channel (short: ENaC) and reabsorb water via water channel Aqp2. ENaC consists of a, b, g subunits and is very important in Na+ balance and blood pressure control. A hormone called aldosterone regulates the production and activity of ENaC at multiple levels including transcription, the first step of ENaC production. However, how aldosterone induces unwinding of DNA that is tightly compacted with a group of proteins called histones remains largely unknown. Our previous studies revealed a novel mechanism regulating aENaC transcription. This mechanism involves a specific enzyme (Dot1a) and Dot1a binding partners Af9 and Af17. Dot1a is produced by Dot1l gene. It can add up to three methyl groups to lysine 79 in histone H3. This modification is referred to H3 K79 methylation. Dot1a-Af9 protein complex reduces aENaC transcription by increasing H3 K79 methylation in the region of DNA that initiates aENaC transcription. Af17 removes the inhibitory effect of Dot1a-Af9 protein complex by competing with Af9 for binding Dot1a and promoting Dot1a export from the nucleus, where transcription occurs. Aldosterone attenuates the inhibitory effect by decreasing the production of Dot1a and Af9 and by reducing Dot1a-Af9 interaction through phosphorylation of Af9. We created Af17-deficient mice to determine whether deletion of Af17 leads to sodium wasting and low blood pressure. Compared with wild-type mice, Af17-deficient mice had lower blood pressure (11 mmHg), higher urine volume, and increased sodium excretion despite mildly increased blood concentrations of aldosterone. Deletion of Af17 led to increased histone H3 K79 methylation and reduced ENaC function. The attenuated function of ENaC resulted from decreased ENaC production and activity. In contrast, inducing high levels of blood aldosterone by a variety of methods completely compensated for Af17 deficiency with respect to sodium handling and blood pressure. Taken together, these data identify Af17 as a potential gene for the maintenance of sodium and blood pressure control and suggest that Af17 has potential as a therapeutic target for the control of blood pressure. We also developed mice lacking Dot1 gene that produces Dot1a in Aqp2-expressing cells. These mice had approximately 20% fewer principal cells and 13%-16% more intercalated cells than control mice. Deletion of Dot1l in principal cells completely eliminated histone H3 K79 methylation in these cells, but unexpectedly, most intercalated cells also had undetectable H3 K79 methylation. These findings suggest that both principal and intercalated cells are offspring of the same Aqp2+ progenitor cells when Dot1l function is disrupted. Using red fluorescence protein as the tracing marker, we not only confirmed that Aqp2+ progenitors give rise to intercalated cells naturally (i.e., without need of Dot1l deletion), but also demonstrated that Aqp2+ progenitors contribute to inter-tubular connection in kidney by differentiating into transitional cells at the junction between the connecting tubule and distal convoluted tubule.

Recent Publications

Publications

Adli M, Parlak M, Li Y, El-Dahr S. Epigenetic States of Nephron Progenitors and Epithelial Differentiation. *J Cell Biochem*. 2015 Jan 5. doi: 10.1002/jcb.25048. [Epub ahead of print] PMID: 25560433

Cuevas CA, Gonzalez AA, Inestrosa NC, Vio CP, Prieto MC. Angiotensin II increases fibronectin and collagen I through the β-catenin dependent signaling in mouse collecting duct cells. *Am J Physiol Renal Physiol*. 2014 Nov 19:ajprenal.00429.2014. doi: 10.1152/ajprenal.00429.2014. [Epub ahead of print]. PMID: 25411386 / PMCID: in Process.

Gonzalez AA, Green T, Luffman C, Bourgeois CR, Gabriel Navar L, Prieto MC. Renal medullary cyclooxygenase-2 and (pro)renin receptor expression during angiotensin II-dependent hypertension. *Am J Physiol Renal Physiol*. 2014 Oct 15;307(8):F962-70. doi: 10.1152/ajprenal.00267.2014. Epub 2014 Aug 20. PMID: 25143455/ PMCID: PMC4200301.

Gonzalez AA, Prieto MC. Renin and the (pro)renin receptor in the renal collecting duct: Role in the pathogenesis of hypertension. *Clin Exp Pharmacol Physiol*. 2015 Jan;42(1):14-21. doi: 10.1111/1440-1681.12319. PMID: 25371190/ PMCID: in Process.

Gonzalez AA, Womack JP, Liu L, Seth DM, Prieto MC. Angiotensin II increases the expression of (pro)renin receptor during low-salt conditions. *Am J Med Sci*. 2014 Nov;348 (5):416-22. doi: 10.1097/MAJ.0000000000000335. PMID: 25250989/ PMCID: in Process.

Miyata K, Satou R, Inui D, Katsurada A, Seth D, Davis A, Urushihara M, Kobori H, Mitchell KD, Navar LG. Renoprotective effects of direct renin inhibition in glomerulonephritis. *Am J Med Sci*. 2014 Oct;348(4):306-14. doi: 10.1097/MAJ.0b013e3182a5b6dd. PMID: 24165783/ PMCID: PMC4000293.

Yan L, Yao X, Bachvarov D, Saifudeen Z, El-Dahr SS. Genome-wide analysis of gestational gene-environment interactions in the developing kidney. *Physiol Genomics*. 2014 Sep 1;46 (17):655-70. doi: 10.1152/physiolgenomics.00035.2014. Epub 2014 Jul 8. PMID: 25005792.

Zhuang Y, Nguyen HT, Burow ME, Zhuo Y, El-Dahr SS, Yao X, Cao S, Flemington EK, Nephew KP, Fang F, Collins-Burow B, Rhodes LV, Yu Q, Jayawickramarajah J, Shan B. Elevated expression of long intergenic non-coding RNA HOTAIR in a basal-like variant of MCF-7 breast cancer cells. *Mol Carcinog*. 2014 Oct 18. doi: 10.1002/mc.22237. [Epub ahead of print]. PMID: 25328122.

Presentations

From August through December, 2014 investigators and physicians affiliated with T.H.R.C.E. participated in many regional, national, & international meetings.

AHA High Blood Pressure Research, San Francisco, CA. Sept. 9-12

- **Navar LG, Shao W, Satou R, Prieto MC, Miyata K, Katsurada A, Mitchell KD.** Increased Renal Angiotensinogen Expression in Non-clipped Kidneys of 2-Kidney 1-Clip Hypertensive Rats. #519.
- **Navar LG, Katsurada A, Fonseca V, Prieto MC, Chalew S, Kobori H**. Augmented Urinary Angiotensinogen in Young Type-1 Diabetic Subjects Correlates with Hemoglobin A1c and Urinary 8-Isoprostane. #559.
- **Park SO, Kirabo A, Baskin R, Seth DM, Navar LG, Fogo AB, Baylis C**. Jak2 Tyrosine Kinase Mediates Angiotensin II Renal Pathogenesis via its Pressor Dependent Actions. #514.
- **Prieto MC, Arita DY, Bourgeois CRT, Satou R.** Hyperglycemia increases Prorenin receptor (PRR) localization at the cell plasma membrane.

ASN Kidney Week 2014, Philadelphia, PA, Nov. 11-16

- **Chen S, Yao X, Saifudeen Z, El-Dahr SS.** HDAC1 and 2 regulates Wnt4 gene expression in the developing kidney. Oral presentation.
- **Hilliard S, Yao X, El-Dahr SS**. The Mdm2-Mdm4-p53 pathway regulates nephron progenitors. Poster presentation.
- **Liu J, El-Dahr SS, Saifudeen Z.** Metabolic Fitness and Self-Renewal of Nephron Progenitor Cells. Poster presentation.
- **Riedl, Song, Yosypiv IV.** Lack of the Prorenin Receptor (PRR) in the Ureteric Bud (UB) Disrupts Developmental Programming of Nephrogenesis. *J Invest Med* 62, A579, 2014.
- **Song R, Preston G, Yosypiv IV**. Collecting duct (CD) prorenin receptor (PRR) contributes to renal sodium reabsorption via regulation of ENaC expression. *ASN on-line publications*, 2014.

Upcoming Scientific Events

February 11-13	AHA American Stroke Association ATVB Meeting; Nashville, TN .
February 26-28	2015 Southern Regional Meeting; New Orleans, Louisiana.
March 13 - 17	ISN World Congress of Nephrology 2015: Cape Town, South Africa.
March 25-26	Tulane University 26th Annual Health Sciences Research Days
March 28 - April 1	Experimental Biology Meeting; Boston, MA.
May 15-19	American Society of Hypertension; New York, New York.

Invited Lectures

THRCE investigators and physicians were invited to lecture at various national and international events.

Hering-Smith, Kathleen:

- "Skipping Stones: Citrate Transport in the Proximal Tubule" on October 13th, 2014 at the Tulane Center for Aging Seminar Series.
- "Skipping Stones: Citrate Transport in the Proximal Tubule" on December 6th, 2014 at the Southern Salt in Sarasota, Florida.

Majid, Dewan S. A:

- "Regulation of inflammatory cytokines in the kidney during high salt intake: implications in salt-sensitive hypertension" on 7th December 2014 at the 4th Biennial Conference of South Asian Association of Physiologists (SAAP) held in Dhaka, Bangladesh.
- Chaired a Plenary Session on "Heavy Metal Toxicity and Oxidative Stress" on 7th December 2014 at the 4th Biennial Conference of SAAP held in Dhaka, Bangladesh.
- "Salt sensitive hypertension: Role of inflammatory cytokines" on 24th December, 2014 at the National Institutes of Cardio-Vascular Diseases (NICVD) in Dhaka, Bangladesh.
- "Why Physiology is important in Medicine?" at the Ibne Sina Medical College, Dhaka, Bangladesh on 24th December, 2014.

Navar, L. Gabriel:

- On September 8, 2014 was the Keynote Speaker at the International Society of Hypertension New Investigator Symposium held in San Francisco, California and presented, "From Mentee to Mentor: Pathways to Emerging Independence."
- "Regulation of Intrarenal Angiotensin II in Normal and Hypertensive Conditions" on September 16, 2014 at the University of California at Merced, California.
- "Urinary Angiotensinogen as an Index of Intrarenal Activation of the Renin-Angiotensin System," on September 23rd, 2014 at the 25th Annual Vascular Biology and Hypertension Symposium held in the University of Alabama at Birmingham, Alabama. Also served as Judge for the Trainee Poster Session.
- "Mechanisms of Intrarenal Angiotensin II Augmentation in Hypertension," on October 9, 2014 at the University of Florida Hypertension Center in Gainesville, Florida.
- The E. Eric Muirhead Lecture, "Activation of the Tubular Renin Angiotensinogen System in Hypertension: Experimental and Translational Studies" on November 5th at the Hypertension Research Day Event held at University of Tennessee Health Science Center, Memphis, Tennessee.

Prieto, Minolfa C:

- Invited on October 22nd 2014 by Prof. Korinna Sanchez to present, "Latin Women in STEM Careers" at L.W. Higgins High School in Marrero, Louisiana.

Calendar of Events

THRCE Seminars

September 11, 2014

NO MEETING
High Blood Pressure Research 2014 Scientific Sessions
San Francisco, California, Sept. 9-12, 2014

September 25, 2014

Sean P. Didion, PhD
Associate Professor, Departments of Pharmacology & Neurology
Director, MD/PhD Program,
The University of Mississippi Medical Center, Jackson, MS.
"Inflammation, Endothelial Dysfunction, and Angiotensin II."

October 9, 2014

No THRCE seminar due to scheduling conflict with the
Tulane Diabetes Research Program special lecture

Speaker: C Ronald Kahn, MD Joslin Diabetes Center and Harvard Medical School
Lecture: Genes environment interactions in the pathogenesis of diabetes and metabolic syndrome

October 23, 2014

Kenneth D. Mitchell, PhD
Professor, Department of Physiology,
Tulane University School of Medicine, New Orleans, LA.
"Renal Derangements in ANG II-Dependent Hypertension: Role of PDG."

November 6, 2014

Aaron S. Dumont, MD
Charles B. Wilson Professor & Chairman, Department of Neurosurgery,
Tulane University School of Medicine, New Orleans, LA.
"Inflammation and Cerebral Aneurysms."

November 20, 2014

Wenzheng Zhang, PhD
Associate Professor, Department of Internal Medicine,
Division of Renal Diseases and Hypertension,
University of Texas Medical School, Houston, TX.
"Epigenetics of Blood Pressure Control and Aqp2+ Renal Progenitor Cells."

December 18 & January 1, 2015

NO MEETING
Winter Holidays

January 15

L. Gabriel Navar, PhD
Chair & Professor, Department of Physiology,
Co-Director, THRCE,
Director, Center of Biomedical Research Excellence in Hypertension & Renal Biology,
Tulane University School of Medicine, New Orleans, LA.
"Translational Studies on Activation of the Intrarenal Renin-Angiotensin System in Type-1 Diabetes."

January 29

Patrick Burgess, MD
Chief Medical Officer, MD Scientific LLC., Charlotte, NC. .
"New Findings from BOSS (Bicarbonate or Saline study)...
A Reduced death related to bicarbonate administration prior to coronary contrast."

February 12

Bysani Chandrasekar, DVM, PhD
Professor, Heart & Vascular Institute
Tulane University School of Medicine, New Orleans, LA .
"RECK and cardiovascular diseases."

Calendar of Events

Continued...

February 26

NO MEETING
Southern Regional Meeting
New Orleans, Louisiana, Sept. 26-28, 2015

Monday, March 9 **

2015 MAYERSON-DILUZIO LECTURE
Jointly Sponsored by THRCE & the Department of Physiology

WALTER F. BORON, MD, PHD
David N. & Inez Myers/Antonio Scarpa Professor & Chairman,
Department of Physiology & Biophysics,
Case Western Reserve University, School of Medicine, Cleveland, OH.
"CO2/HCO3− sensing in the renal proximal tubule."

Wed., March 11 **

MEDICINE GRAND ROUNDS
Jointly Sponsored by THRCE & the Department of Medicine, Division of Nephrology

DAVID G. HARRISON, MD
Betty and Jack Bailey Professor of Medicine and Pharmacology,
Director of Clinical Pharmacology, Vanderbilt University, Nashville, TN.
"The History of Hypertension and the Mosaic Theory."

March 12
Time: 8:30am-9:30am

Special Seminar: WORLD KIDNEY DAY 2015
Jointly Sponsored by THRCE & the Department of Physiology

DAVID G. HARRISON, MD
Betty and Jack Bailey Professor of Medicine and Pharmacology,
Director of Clinical Pharmacology,
Vanderbilt University, Nashville, TN.
"Inflammation, Immunity and Hypertension."

March 26

HENRY A PUNZI, MD
Internist, Punzi Medical Center and Trinity Hypertension Research Institute,
Carrollton, TX.
"Hypertension: How low should we go."

April 9

NAZIH L NAKHOUL, PHD
Research Associate Professor, Departments of Medicine & Physiology,
Tulane University School of Medicine, New Orleans, LA.
TBA

April 23

KAFAIT U. MALIK, PHD
Professor of Pharmacology,
College of Medicine, University of Tennessee Health Science Center,
Memphis, TN.
"Contribution of Cytochrome P450 1B1 to sex differences in the development of hypertension and its pathogenesis."

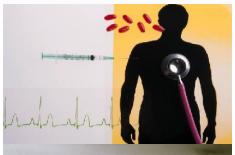
Conferences are held alternative Thursdays at 4:00pm in the Tulane Medical School, Pharmacology Library, Room 4700
***** Denotes the seminar date is not our normally scheduled day.***

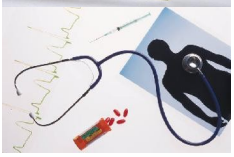
CORE Facilities & Services

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 in hypertension 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**: This facility 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:** This facility maintains and generates new breeding pairs, does 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/.**





T.H.R.C.E.

Tulane Hypertension And Renal Center of Excellence will appreciate any support for the continual development of the center, the publication of the THRCE newsletters, and the support of the THRCE bi-weekly seminars series. Any 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 Program Coordinator, Nina 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.